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A cohort study of dietary fibre intake and menarche

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Abstract

Objective: To evaluate the influence of dietary fibre on menarche in a cohort of pre-menarcheal girls.
Design: Prospective cohort study.
Setting: Ontario, Canada.
Subjects: Free-living pre-menarcheal girls ($n = 637$), 6 to 14 years of age.
Methodology: Information on dietary intake, physical activity and date of menarche was collected at baseline and was updated annually by self-administered questionnaires for three years. Cox proportional hazards models were used to evaluate the association between dietary fibre and menarche, adjusting for age at entry to the study and potential confounders.
Results: A higher intake of energy-adjusted dietary fibre was associated with a lower risk of (i.e. a later age at) menarche (relative hazard 0.54, 95% confidence interval (CI) 0.31–0.94 for highest vs. lowest quartile, P for trend = 0.027). At the fibre component level, a higher intake of energy-adjusted cellulose was associated with a lower risk of menarche (relative hazard 0.45, 95% CI 0.26–0.76, P for trend = 0.009).
Conclusions: The findings are consistent with the hypothesis that pre-menarcheal dietary intake can influence menarche.

Keywords
Menarche
Dietary fibre
Cellulose
Monounsaturated fatty acids
Puberty
Breast cancer
Risk factors

Understanding the predictors of menarche is important because of its association with a number of health outcomes such as breast cancer[1–3], endometrial cancer[4], pancreatic cancer[5], coronary heart disease[6], osteoporosis[7], Hashimoto's thyroiditis[8], rheumatoid arthritis[9] and unsuccessful reproductive events[10]. Various anthropometric, socio-economic, familial, nutritional and lifestyle predictors have been studied in relation to menarche[11–14]. The most consistent finding has been the inverse association of weight and height with age at menarche[15]. Available studies are less clear regarding the relation with other factors, particularly nutritional factors. Results from an international ecological study showed a significant inverse correlation between intakes of dietary fibre and age at menarche[16]. A few prospective studies also reported significant associations between age at menarche and intakes of either dietary fibre[17] or foods with high fibre content[18]. Besides dietary fibre, higher intakes of monounsaturated fatty acids, vitamin A, iron and thiamine have also been found to be associated with a later age at menarche[19,20]. On the other hand, intake of dietary fat has not been found to be associated with menarche[21,22].

There are a number of plausible biological mechanisms by which dietary fibre might influence age at menarche, including alteration in enterohepatic circulation of oestrogen[23–25], reduction of bioavailability of oestrogen[26,27], and suppression of gonadotrophin production[17]. Therefore, in this study, we evaluated the influence of dietary fibre on menarche in a cohort of girls in Ontario, Canada. Previous research in this area has been based largely on the use of food records for the measurement of dietary intake[19–21]. Food records do not measure habitual intake, and therefore, in order to address this limitation, we used a food-frequency questionnaire designed to measure habitual dietary intake in girls[28].

Methods

In September 1992, a cohort of girls between the ages of 6 and 14 years who had not attained menarche was identified from the daughters of adults who were participating in a cohort study focusing on diet and cancer risk. These adults were recruited by volunteers from the Canadian Cancer Society. Of the 763 daughters identified in the adult cohort, 637 were eligible for the study because they had not yet experienced menarche. Starting in 1993

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(the first follow-up), packages containing self-administered semi-quantitative food-frequency and lifestyle questionnaires were sent to the participants approximately every twelve months. All questionnaires and communications were addressed to the mothers to ensure a systematic and reliable return of questionnaires. Menarcheal status and the timing of menarche were ascertained from the responses to the following questions in the lifestyle questionnaire: 'Has she started her menstrual periods yet?' and 'In which month and year did her menstrual periods start?'. Maternal age of menarche (in years) was obtained from questionnaires in the cohort of adults. Reminder letters and reminder packages that included reminder letters and new sets of questionnaires were sent to non-respondents following a planned schedule at each of the annual follow-ups. Phone interviews were also conducted during the third follow-up to ascertain information on menarche. The follow-up was terminated 1100 days from the date of reporting on the first returned questionnaire, which was 7 April 1996. The study protocol was approved by the human ethics committee of the University of Toronto.

Food-frequency questionnaire

A 162-item food-frequency questionnaire[29,30] was used in this study to estimate the participants' usual nutrient and energy intake over the previous year. On each occasion, the participants were asked to report their usual frequency of consumption and the usual portion size of foods over the past twelve months. The frequencies were reported as the number of times per month, week or day that a food item was consumed. Participants reported their usual portion size as a multiple of the standard portion size by reference to the food model photographs.

Reproducibility and validity of the food-frequency questionnaire were studied in a sample of girls in Ontario between the ages of 7 and 12 years[28]. The test–retest reproducibility and the correlation between fibre intake measured using the questionnaire and the mean of 14 days of food record data were amongst the highest for all nutrients measured in that study, with Pearson correlation coefficients of 0.71 (95% confidence interval (CI) 0.58–0.80) and 0.56 (95% CI 0.38–0.70), respectively.

Lifestyle and physical activity questionnaire

The lifestyle questionnaire collected information on anthropometry and on physical activity using a multi-item habitual physical activity questionnaire[31]. The latter was used to generate estimates of energy expenditure.

Statistical analysis

The nutrient intakes from the dietary questionnaires were estimated using a food database developed previously[29]. This database was originally based on *USDA Handbook No. 8* (US Department of Agriculture, 1999), but was expanded to incorporate nutrient values for Canadian foods and to include values for several nutrients not present in the USDA database. Values for components of the dietary fibre that were not originally available in the database were obtained from the tables of McCance and Widdowson[32–34] with standardisation based on the carbohydrate content of the food items.

For the analyses reported here, participants were categorised according to their quartile of energy-adjusted nutrient intake[35]. We used Cox proportional hazards regression to examine the independent association of dietary fibre and its components with menarche in the presence of covariates, with follow-up time as the underlying time metric[36]. Subjects who remained premenarcheal at the end of the follow-up, did not respond to the questionnaires, had withdrawn from the study, or who had moved and were unable to be traced at the time of follow-up were treated as censored observations. In the basic multivariate analyses, we adjusted for age at entry (a continuous variable), time-dependent age at entry, body weight, birth weight, log energy intake, and maternal age at menarche. The variable 'time-dependent age at entry' was calculated by multiplying the 'age at entry' by the difference between the natural logarithm of the study time variable 'time at entry' and the mean value of the logarithms of 'time at entry'. In addition, other potential confounding nutrient factors were evaluated in the full multivariate analyses based on the 10% change-in-coefficient method[37]. The potential confounding effect of socio-economic status was evaluated by using paternal occupational prestige rating. Paternal occupation was first coded into Standard Occupational Classification[38] and then coded into Occupational Prestige Ratings[39] to calculate paternal occupational prestige rating.

Interactions between variables, the presence of influential observations and the proportional hazards assumption were tested[40,41]. All significance tests were two-tailed, and the level of significance was set at 5%. The analyses were performed with the SAS statistical package version 6.12 (SAS Institute, Cary, NC). Tests of linear trend for the quartiles were performed by converting each set of quartile categories of the independent variables into a graded continuous variable (with value of 0, 1, 2 or 3) and assessing the statistical significance associated with this variable in Cox model using the Wald χ^2 test[36].

Results

Of the 589 subjects who had complete information on their exposure and menarche, 187 experienced menarche during the follow-up period. Forty-eight subjects were excluded from the data analysis because they either never responded to any of the follow-up mailings ($n = 28$) or did not provide information on their date of menarche ($n = 20$). The response rates at the end of the first, second and third follow-ups were 85.6% (545/637), 82.1% (478/582) and 97.5% (583/598), respectively. The median

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Table 1 Baseline characteristics of study subjects by status of menarche at the end of the follow-up

Characteristics	Menarche		Non-menarche	
	Number	Median (min, max)	Number	Median (min, max)
Age at entry into study (years)	187*	11.3 (7.0, 13.2)	402	9.1 (6.2, 13.6)
Weight (kg)	181	41 (26, 73)	379	30 (17, 85)
Height (m)	182	1.5 (1.0, 1.7)	380	1.4 (1.1, 1.7)
Body mass index (kg m^{-2})	179	18.3 (13.2, 40.6)	365	16.5 (12.2, 38.0)
Birth weight (kg)	181	3.5 (1.9, 5.1)	391	3.4 (1.6, 6.4)
Energy expenditure (kcal week^{-1})	181	4736 (194, 17946)	379	3326 (97, 18674)
Maternal age of menarche (years)	114	12 (9, 16)	242	13 (9, 17)
Paternal occupational prestige rating	187	50.6 (23.3, 86.1)	402	50.6 (22.3, 86.1)
Total energy intake (kcal day^{-1})	187	2432 (1163, 5886)	402	2336 (763, 5773)
Dietary protein (g day^{-1})	187	94 (42, 190)	402	94 (25, 314)
Dietary fat (g day^{-1})	187	95 (38, 204)	402	89 (26, 263)
Carbohydrate (g day^{-1})	187	302 (136, 888)	402	296 (97, 794)
Dietary fibre (g day^{-1})	187	22.4 (8.2, 55.1)	402	21.5 (4.8, 87.2)
Soluble fibre (g day^{-1})	187	6.0 (2.2, 15.3)	402	5.6 (1.4, 18.8)
Insoluble fibre (g day^{-1})	187	4.2 (1.3, 13.6)	402	4.3 (0.8, 21.0)
Cellulose (g day^{-1})	187	4.8 (1.5, 15.4)	402	4.9 (0.9, 28.0)
Lignin (g day^{-1})	187	1.7 (0.4, 4.5)	402	1.6 (0.3, 8.3)
Resistant starch (g day^{-1})	187	0.6 (0.1, 2.1)	402	0.7 (0.2, 4.2)
Animal fat (g day^{-1})	187	60.1 (16.6, 135.1)	402	58.6 (17.6, 232.9)
Vegetable fat (g day^{-1})	187	32.7 (6.6, 99.0)	402	30.5 (2.8, 146.6)
Saturated fatty acids (g day^{-1})	187	36.9 (13.8, 82.2)	402	34.2 (11.4, 101.4)
Monounsaturated fatty acids (g day^{-1})	187	39.1 (16.4, 78.1)	402	37.4 (10.4, 105.4)
Polyunsaturated fatty acids (g day^{-1})	187	10.5 (2.7, 29.2)	402	9.3 (1.4, 47.6)
Cholesterol (mg day^{-1})	187	320.4 (113.4, 840.8)	402	313.6 (104.7, 1279.8)

*Numbers vary due to missing values for some variables.

length of follow-up was 863 days with a range of 13 to 1100 days.

The baseline characteristics of the participants are shown in Table 1. The median age of all subjects at recruitment was 9.7 years (range 6.2 to 13.6 years), with subjects who experienced menarche during the study period being older than those who did not by 2.2 years. The age at menarche ranged from 8.5 to 15.6 years with a median age of 13.6 years.

Results of the univariate analyses of the association between menarche and various anthropometric variables are presented in Table 2. Height, weight and body mass index showed strong direct associations with menarche, while birth weight and maternal age at menarche had strong inverse associations with menarche. In other words, a greater birth weight and later maternal age at menarche are associated with a later age at menarche in daughters.

In multivariate analyses (Table 3), a relatively high intake of energy-adjusted total dietary fibre was significantly associated with reduced risk of menarche (in other words, with a later age at menarche). When risk was examined by components of fibre, higher intakes of energy-adjusted cellulose and lignin were significantly associated with reduced risk of menarche. The hazard ratios for lignin became statistically non-significant with the addition of the cellulose variable to the same model, whereas the cellulose variable remained significant with the inclusion of lignin. This indicated that cellulose, unlike lignin, was independently associated with menarche.

Increased intake of energy-adjusted animal fat was also inversely associated with menarche. Of the four fat components studied, energy-adjusted monounsaturated fatty acids showed the strongest association with menarche.

All questionnaires were addressed to the mothers to ensure a systematic and reliable return of questionnaires. The effect of who completed the questionnaires on the association between diet and menarche was evaluated by including an indicator variable of whether the questionnaires were completed by the parents with or without the assistance of their daughters in the model. The indicator variable was not significant ($P = 0.55$), indicating that observed association between diet and menarche did not depend on whether the questionnaires were completed by the parents alone or with the assistance of their daughters.

Discussion

In this prospective study of Ontario girls, we observed a statistically significant inverse association between energy-adjusted dietary fibre intake and risk of menarche. Risk of menarche in the highest quartile group of dietary fibre intake (>25.5 g day^{-1}) was approximately half that for girls in the lowest quartile group of intake (≤18.2 g day^{-1}). The association was not observed for all fibre fractions and this is not surprising, since dietary fibre is a heterogeneous group of substances with different solubility in water and different three-dimensional structures that may have distinct effects on oestrogen metabolism[42,43].

There are a few published epidemiological reports of the relation between intakes of dietary fibre and menarche. An ecological study of 46 countries and

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Table 2 Age-adjusted relative hazards for menarche by levels of selected variables

Variable	Quartile	Relative hazard*	*P* (trend)
Height (m)	≤1.32	1.00 (referent)	0.0002
	>1.32 to ≤1.40	1.14 (0.60–2.20)†	
	>1.40 to ≤1.48	1.78 (0.97–3.28)	
	>1.48	2.81 (1.47–5.36)	
Weight (kg)	≤28	1.00 (referent)	0.0001
	>28 to ≤34	2.16 (1.02–4.57)	
	>34 to ≤41	3.83 (1.84–7.97)	
	>41	6.88 (3.28–14.41)	
Body mass index (kg m^{-2})	≤15.62	1.00 (referent)	0.0002
	>15.62 to ≤17.09	2.71 (1.61–4.59)	
	>17.09 to ≤19.41	2.72 (1.63–4.55)	
	>19.41	2.98 (1.78–4.99)	
Birth weight (kg)	≤3.20	1.00 (referent)	0.0033
	>3.20 to ≤3.46	0.71 (0.47–1.07)	
	>3.46 to ≤3.83	0.61 (0.41–0.90)	
	>3.83	0.56 (0.37–0.83)	
Energy expenditure (kcal week^{-1})	≤1943.6	1.00 (referent)	0.0389
	>1943.6 to ≤3724.9	1.10 (0.69–1.74)	
	>3724.9 to ≤6191.3	1.45 (0.93–2.27)	
	>6191.3	1.46 (0.95–2.24)	
Maternal age of menarche (years)	≤12	1.00 (referent)	0.0003
	13	1.01 (0.69–1.49)	
	14	0.78 (0.53–1.15)	
	>14	0.39 (0.24–0.64)	
Paternal occupational prestige rating	≤39.64	1.00 (referent)	0.2809
	>39.64 to ≤50.64	1.22 (0.82–1.80)	
	>50.64 to ≤64.19	0.81 (0.51–1.28)	
	>64.19	1.43 (0.96–2.14)	

* Adjusted for age at entry into study.
† Numbers in parentheses are 95% confidence intervals.

communities found a significant positive correlation ($r = 0.84$) between dietary fibre (g/1000 kcal) and age of menarche[16]. In a prospective study on 62 girls, girls with the highest daily intake of dietary fibre had slower sexual development during puberty[17]. Dietary fibre was found to be a significant factor associated with girls who had attained menarche at age 14.3 years when energy intake, height, timing of the 7-day food record, vegetarian protein and polysaccharides were adjusted in the multiple regression analysis. Another cross-sectional study on 777 girls in Spain reported that later age of menarche was significantly associated with an increase intake of nuts and seeds in girls who were over 13 years old[18]. The authors suggested that dietary fibre might be one of the underlying nutrients because of their high contents in nuts and seeds. However, in a nested case–control study of 666 girls conducted in Canada, age- and energy-adjusted crude dietary fibre was not significantly associated with menarche (odds ratio (OR) 0.8, 95% CI 0.5–1.3 for highest vs. lowest quartile level of intake)[19]. A similar result (relative risk 0.9, 95% CI 0.7–1.1 for highest vs. lowest quartile level of intake) was obtained when the data from the full cohort of 2299 subjects were analysed[44].

Early pubertal plasma oestrogen levels are predictors of the rate of pubertal development towards menarche[45]. Increased dietary fibre intake may reduce the availability of circulating oestrogen and thereby influence pubertal development and menarche. Several mechanisms have been proposed for an effect of dietary fibre on oestrogen metabolism, including reduced deconjugation of oestrogen conjugates[23] and hence reduced uptake of free oestrogen via the enterohepatic circulation; increased faecal oestrogen excretion by binding of deconjugated oestrogen[24,25]; reduced bioavailability of oestradiol due to increased hepatic sex hormone-binding globulin levels (SHBG)[26]; competition with oestrogen receptors in oestrogen-sensitive tissues[27]; and reduced gonadotrophin production through direct action on the hypothalamic–pituitary–gonadal system[17].

We also observed an inverse association between energy-adjusted intake of monounsaturated fatty acids and menarche. Similar associations have been reported in previous studies. Moisan *et al.*[19] reported a statistically significant inverse association between menarche and age- and energy-adjusted monounsaturated fatty acids in their case–control study of 333 Canadian girls (OR 0.6, 95% CI 0.4–0.9 for highest vs. lowest quartile level of intake). In a later report from the same group of investigators, a higher intake of monounsaturated fatty acids was associated with an earlier menarche (relative risk 1.3, 95% CI 1.1–1.5 for highest vs. lowest quartile)[44]. Animal fat was found to be inversely associated with menarche in this study, with a relative hazard of 0.52 (95% CI 0.32–0.85) for individuals with an energy-adjusted animal fat intake of above 66.4 g day^{-1} compared with those with intakes equal to or below 50.3 g day^{-1}. The

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Table 3 Multivariate relative hazards for menarche by total energy intake, energy-adjusted dietary fibre, dietary fat and their components

Variable	Quartile	Relative hazard	*P* (trend)
Total energy intake (kcal day^{-1})	≤1980.3*	1.00 (referent)	0.5823
	>1980.3 to ≤2362.1	1.47† (0.95–2.28)‡	
	>2362.1 to ≤2798.8	1.25 (0.81–1.92)	
	>2798.8	1.21 (0.79–1.86)	
Dietary fibre (g day^{-1})	≤18.19	1.00 (referent)	0.0269
	>18.19 to ≤21.81	0.89§ (0.55–1.44)	
	>21.81 to ≤25.48	0.81 (0.50–1.31)	
	> 25.48	0.54 (0.31–0.94)	
Soluble fibre (g day^{-1})	≤4.78	1.00 (referent)	0.3451
	>4.78 to ≤5.73	0.85 (0.53–1.35)	
	>5.73 to ≤6.82	0.91 (0.55–1.52)	
	>6.82	0.71 (0.39–1.30)	
Insoluble fibre (g day^{-1})	≤3.30	1.00 (referent)	0.0455
	>3.30 to ≤4.19	1.19 (0.77–1.84)	
	>4.19 to ≤5.29	0.92 (0.58–1.46)	
	>5.29	0.60 (0.36–1.01)	
Cellulose (g day^{-1})	≤3.75	1.00 (referent)	0.0090
	>3.75 to ≤4.86	0.62 (0.40–0.98)	
	>4.86 to ≤6.11	0.70 (0.43–1.12)	
	>6.11	0.45 (0.26–0.76)	
Lignin (g day^{-1})	≤1.29	1.00 (referent)	0.0269
	>1.29 to ≤1.63	0.66 (0.42–1.03)	
	>1.63 to ≤2.03	0.72 (0.46–1.13)	
	>2.03	0.52 (0.32–0.86)	
Resistant starch (g day^{-1})	≤0.53	1.00 (referent)	0.1416
	>0.53 to ≤0.68	0.68 (0.46–1.02)	
	>0.68 to ≤0.85	0.77 (0.49–1.21)	
	>0.85	0.70 (0.44–1.10)	
Animal fat (g day^{-1})	≤50.28	1.00 (referent)	0.0313
	>50.28 to ≤58.30	0.48 (0.30–0.74)	
	>58.30 to ≤66.36	0.48 (0.30–0.78)	
	>66.36	0.52 (0.32–0.85)	
Vegetable fat (g day^{-1})	≤25.82	1.00 (referent)	0.3527
	>25.82 to ≤31.59	0.73 (0.46–1.16)	
	>31.59 to ≤37.09	0.95 (0.60–1.50)	
	>37.09	1.12 (0.72–1.76)	
Saturated fatty acids (g day^{-1})	≤31.03	1.00 (referent)	0.3974
	>31.03 to ≤34.62	0.60 (0.37–0.98)	
	>34.62 to ≤38.32	0.72 (0.41–1.24)	
	>38.32	0.63 (0.34–1.18)	
Monounsaturated fatty acids (g day^{-1})	≤34.68	1.00 (referent)	0.1773
	>34.68 to ≤37.77	0.54 (0.33–0.86)	
	>37.77 to ≤41.10	0.59 (0.35–1.00)	
	>41.10	0.59 (0.33–1.07)	
Polyunsaturated fatty acids (g day^{-1})	≤7.86	1.00 (referent)	0.1497
	> 7.86 to ≤9.56	1.19 (0.76–1.85)	
	>9.56 to ≤11.71	1.09 (0.69–1.71)	
	>11.71	1.44 (0.93–2.24)	
Cholesterol (mg day^{-1})	≤272.32	1.00 (referent)	0.2885
	>272.32 to ≤316.28	0.60 (0.38–0.96)	
	>316.28 to ≤357.03	0.71 (0.43–1.17)	
	>357.03	0.65 (0.37–1.14)	

* Cut-off points for quartiles are exponentiated to improve interpretability.
† Adjusted for age at entry into study (continuous), time-dependent age at entry into study (continuous), body weight (≤28, >28 to ≤34, >34 to ≤41 or >41 kg), maternal age at menarche (≤12, 13, 14 or >14 years) and birth weight (≤3.20, >3.20 to ≤3.46, >3.46 to ≤3.83 or >3.83 kg).
‡ Numbers in parentheses are 95% confidence intervals.
§ Adjusted for age at entry into study (continuous), time-dependent age at entry into study (continuous), body weight (≤28, >28 to ≤34, >34 to ≤41 or >41 kg), maternal age at menarche (≤12, 13, 14 or >14 years), birth weight (≤3.20, >3.20 to ≤3.46, >3.46 to ≤3.83 or >3.83 kg), log energy intake (quartile) and nutrient confounding variables.

significant inverse association between animal fat and menarche is not surprising since the top contributing source of monounsaturated fat intake is food of animal origin, which includes roast beef, roast pork, pork chops, steak and baked ham. Energy intake was not associated with menarche in our study, similar to the results from other studies[14,21,46]. The association between monounsaturated fat and menarche is intriguing and deserves further study given the interrelation among monounsaturated fat, menarche and breast cancer risk. The incidence

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rates of breast cancer observed in Mediterranean countries are about half that in the United States[47] and these Mediterranean countries also have higher average intakes of monounsaturated fat than other countries owing to the use of olive oil as their primary source of fat[48,49]. Approximately 29% of daily caloric intake are derived from monounsaturated fat in these countries[50]. In Canada, the main source of monounsaturated fat is from canola oil, which currently accounts for about three-quarters of all vegetable oils processed. Canola oil consists of 61% monounsaturated fat and is second to olive oil in terms of content (75%)[51]. Cohort studies[52,53], case–control studies[54,55] and animal studies[56] have also observed a protective effect of monounsaturated fat on breast cancer risk. Assuming that dietary patterns are reasonably stable throughout life, it is plausible that the effect of monounsaturated fat on breast cancer risk is mediated indirectly through an influence on the timing of menarche.

The results for non-nutritional variables were in agreement with those reported in other published studies (Table 2). Weight was positively associated with menarche, with a relative hazard of 6.88 (95% CI 3.28–14.41) for individuals over 41 kg compared with those weighing 28 kg or less. Maclure *et al.*[46] reported a lower hazard ratio of 2.2 for individuals over 40 kg relative to those under 25 kg. Moisan *et al.*[44] also found that body weight was significantly associated with menarche, with relative hazard of 5.8 (95% CI 4.7–7.2) between the highest and lowest quartiles. Our study also found an inverse association between maternal age at menarche and the risk of their daughters' menarche. Individuals with mothers whose menarche occurred after age 14 years had a risk 0.39 times that of those with maternal menarche at 12 years of age or earlier. Petridou *et al.*[3] found that maternal age at menarche was inversely associated with daughter's age at menarche with a relative hazard of 0.82 (95% CI 0.73–0.92) for each one-year increase in maternal age at menarche. Socio-economic status, as reflected by the paternal occupational prestige rating, was not associated with menarche in our study population. This observation is not surprising since the difference in dietary quality across socio-economic groups in the North American population is narrower today[57] than it was in the 1960s, when studies that reported a strong association between socio-economic status and menarche were conducted[58]. Birth weight was inversely associated with menarche (relative hazard 0.56, 95% CI 0.37–0.83 for highest vs. lowest quartile). This is in agreement with another study in which girls who were heavier at birth (highest quintile) had a later age of menarche by an average of 2.2 months compared with those who were in the lowest quintile[59].

The use of a prospective design and annual follow-up of the cohort in our study reduced the possibility of recall bias. The completeness of the follow-up and adjustment for a wide range of potentially relevant confounding variables also strengthen our results. In addition, our study is the first one to focus on components of dietary fibre. The lack of association between dietary fibre and menarche in previous studies may be attributed, in part, to the fact that dietary fibre was considered as a single entity. A major limitation of the present study is that the length of the follow-up period was relatively short. Of the 589 subjects included in the analyses, 402 of them had not reached menarche at the end of the follow-up. Reducing the number of censored observations by a longer follow-up period could increase study power and minimise the possibility of follow-up bias arising from insufficient duration of follow-up[60]. In addition, it is possible that participants had been misclassified for dietary variables because of measurement error. However, for polytomous exposures, the effect of non-differential exposure misclassification always results in attenuation of associations when comparing the lowest and highest levels of risk[61]. Therefore, the relative hazards of menarche for the highest versus lowest quartile were unlikely to be overestimated. Although misclassification arising from categorisation of covariates is possible, misspecification of the model is also a concern if the nutrients were treated as continuous variables. The assumption of a linear relationship between dietary variables and menarche is unlikely to be met because most dietary factors have limits in absorption, transport and metabolism. Nevertheless, similar results were observed for the inverse association between menarche and dietary fibre, cellulose and monounsaturated fat when these nutrients were treated as continuous variables. Furthermore, the reported median age at menarche is higher than that reported in most epidemiological studies. However, the proper approach for estimating the age at menarche of a population is to employ probit analysis from data collected by the *status quo* method, in which subjects from a representative sample of the population are asked for their precise age and whether they have reached menarche. The prospective method used in this study is suitable for revealing the age at menarche at the individual level. Since the study was terminated while 68% of the participants still had not reached menarche and the median age at that time was 12.2 years, it is likely that the median age at menarche would ultimately be lower than the observed 13.6 years if the study cohort were followed for longer. Nevertheless, this does not affect the internal validity of the results.

In summary, we found that a high dietary fibre intake is associated with a later age at menarche in this prospective study. From a public health perspective, a diet consisting of a variety of foods that provide a natural source of dietary fibre (e.g. grain products, vegetables and fruit) is compatible with the postulated beneficial roles of dietary fibre in promoting normal bowel function[62] and preventing chronic diseases[63]. Although the ideal dietary fibre intake has not been defined, it has been recommended that the dietary fibre intake of children older than 2 years be increased to an amount equal to or greater than their

age plus 5 g per day, with the ultimate aim of achieving intakes of 25–35 g day^{-1} after the age of 20 years[64].

References

1 Pike MC, Karilo MD, Henderson BE, Casagrande JT, Hoel DG. 'Hormonal' risk factors, 'breast tissue age' and the age-incidence of breast cancer. *Nature* 1983; **303**: 767–70.
2 Kelsey JL, Hildreth NG *Breast and Gynecologic Cancer Epidemiology*. Boca Raton, FL: CRC Press, 1983; 5–70.
3 Petridou E, Syrigou E, Toupadaki N, Zavitsanos Z, Willett W, Trichopoulos D. Determinants of age at menarche as early life predictors of breast cancer risk. *Int. J. Cancer* 1996; **68**: 193–8.
4 Brinton LA, Berman ML, Mortel R, Twiggs LB, Barrett RJ, Wilbanks GD, Lannom L, Hoover RN. Reproductive, menstrual, and medical risk factors for endometrial cancer: results from a case–control study. *Am. J. Obstet. Gynecol.* 1992; **167**: 1317–25.
5 Kalapothaki V, Tzonou A, Hsieh CC, Toupadaki N, Karakatsani A, Trichopoulos D. Tobacco, ethanol, coffee, pancreatitis, diabetes mellitus, and cholelithiasis as risk factors for pancreatic carcinoma. *Cancer Causes Control* 1993; **4**: 375–82.
6 Colditz GA, Willett WC, Stampfer MJ, Rosner B, Speizer FE, Hennekens CH. A prospective study of age at menarche, parity, age at first birth, and coronary heart disease. *Am. J. Epidemiol.* 1987; **126**: 861–70.
7 Matkovic V, Jelic T, Wardlaw GM, Ilich JZ, Goel PK, Wright JK, Andon MB, Smith KT, Heaney RP. Timing of peak bone mass in caucasian females and its implication for the prevention of osteoporosis. Inference from a cross-sectional study. *J. Clin. Invest.* 1994; **93**: 799–808.
8 Phillips DI, Lazarus JH, Butland BK. The influence of pregnancy and reproductive span on the occurrence of autoimmune thyroiditis. *Clin. Endocrinol.* 1990; **32**: 301–6.
9 Deighton CM, Sykes H, Walker DJ. Rheumatoid arthritis, HLA identity, and age at menarche. *Ann. Rheum. Dis.* 1993; **52**: 322–6.
10 Sandler DP, Wilcox AJ, Horney LF. Age at menarche and subsequent reproductive events. *Am. J. Epidemiol.* 1984; **119**: 765–74.
11 Jenicek M, Demirjian A. Age at menarche in French Canadian urban girls. *Ann. Hum. Biol.* 1974; **1**: 339–46.
12 Frisch RE. Fatness and fertility. *Sci. Am.* 1988; **258**: 88–95.
13 Malina RM, Ryan RC, Bonci CM. Age at menarche in athletes and their mothers and sisters. *Ann. Hum. Biol.* 1994; **21**: 417–22.
14 Moisan J, Meyer F, Gingras S. Leisure physical activity and age at menarche. *Med. Sci. Sports Exerc.* 1991; **23**: 1170–4.
15 Koprowski C, Ross RK, Mack WJ, Henderson BE, Bernstein L. Diet, body size and menarche in a multiethnic cohort. *Br. J. Cancer* 1999; **79**: 1907–11.
16 Hughes RE, Jones E. Intake of dietary fiber and the age of menarche. *Ann. Hum. Biol.* 1985; **12**: 325–32.
17 De Ridder CM, Thijssen JHH, Van't Veer, Duurn RV, Bruning PF, Zonderland ML, Erich WBM. Dietary habits, sexual maturation, and plasma hormones in pubertal girls: a longitudinal study. *Am. J. Clin. Nutr.* 1991; **54**: 805–13.
18 Soriguer FJ, Gonzalez-Romero S, Esteva I, Garcia-Arnés JA, Tinahones F, Ruiz De Adana MS, Olveira G, Mancha I, Vazques F. Does the intake of nuts and seeds alter the appearance of menarche? *Acta Obstet. Gynecol. Scand.* 1995; **74**: 455–61.
19 Moisan J, Meyer F, Gingras S. A nested case–control study of the correlates of early menarche. *Am. J. Epidemiol.* 1990; **132**: 953–61.
20 Kissinger DG, Sanchez A. The association of dietary factors with the age of menarche. *Nutr. Res.* 1987; **7**: 471–9.
21 Meyer F, Moisan J, Marcoux D, Bouchard C. Dietary and physical determinants of menarche. *Epidemiology* 1990; **1**: 377–81.
22 Merzenich H, Boeing H, Wahrendorf J. Dietary fat and sports activity as determinants for age at menarche. *Am. J. Epidemiol.* 1993; **138**: 217–24.
23 Goldin BR, Adlercreutz H, Gorbach SL, Warram JH, Dwyer JT, Swenson L, Woods MN. Estrogen excretion patterns and plasma levels in vegetarian and omnivorous women. *N. Engl. J. Med.* 1982; **307**: 1542–7.
24 Shultz TD, Howie BJ. In vitro binding of steroid hormones by natural and purified fibers. *Nutr. Cancer* 1986; **8**: 141–7.
25 Arts CJM, Govers CARL, Van den Berg H, Wolters MGE, Van Leeuwen P, Thijssen JHH. In vitro binding of estrogens by dietary fiber and the in vivo apparent digestibility tested in pigs. *J. Steroid Biochem. Mol. Biol.* 1991; **38**: 621–8.
26 Adlercreutz H, Höckerstedt K, Bannwart C, Bloigu S, Hämäläinen E, Fotsis T, Ollus A. Effect of dietary components, including lignans and phytoestrogens, on enterohepatic circulation and liver metabolism of estrogens and on sex hormone binding globulin (SHBG). *J. Steroid Biochem.* 1987; **27**: 1135–44.
27 Setchell KDR, Adlercreutz H. Mammalian lignans and phyto-oestrogens. Recent studies on their formation, metabolism and biological role in health and disease. In: Rowland IR, ed. *Role of the Gut Flora in Toxicity and Cancer*. London: Academic Press, 1988; 315–45.
28 Arnold JE, Rohan T, Howe G, Leblanc M. Reproducibility and validity of a food-frequency questionnaire designed for use in girls age 7 to 12 years. *Ann. Epidemiol.* 1995; **5**: 369–77.
29 Jain M, Harrison L, Howe GR, Miller AB. Evaluation of a self-administered dietary questionnaire for use in a cohort study. *Am. J. Clin. Nutr.* 1982; **36**: 931–5.
30 Jain M, Howe GR, Rohan T. Dietary assessment in epidemiology: comparison of a food frequency and a diet history questionnaire with a 7-day food record. *Am. J. Epidemiol.* 1996; **143**: 953–60.
31 Koo MM, Rohan TE. Comparison of four habitual physical activity questionnaires in girls aged 7–15 yr. *Med. Sci. Sports Exerc.* 1999; **31**: 421–7.
32 Holland B, Unwin ID, Buss DH. *Cereals and Cereal Products. Third Supplement to McCance & Widdowson's The Composition of Foods*. The Royal Society of Chemistry & Ministry of Agriculture, Fisheries and Food. London: HMSO, 1988.
33 Holland B, Unwin ID, Buss DH. *Vegetables, Herbs, and Spices. Fifth Supplement to McCance & Widdowson's The Composition of Foods*. The Royal Society of Chemistry & Ministry of Agriculture, Fisheries and Food London: HMSO, 1991.
34 Holland B, Unwin ID, Buss DH. *Fruit and Nuts. First Supplement to McCance & Widdowson's The Composition of Foods*, 5th ed. The Royal Society of Chemistry & Ministry of Agriculture, Fisheries and Food. London: HMSO, 1992.
35 Willett W, Stampfer MJ. Total energy intake: implications for epidemiologic analyses. *Am. J. Epidemiol.* 1986; **124**: 17–27.
36 Cox DR, Oakes D. *Analysis of Survival Data*. New York: Chapman & Hall, 1984.
37 Maldonado G, Greenland S. Simulation study by confounder-selection strategies. *Am. J. Epidemiol.* 1993; **138**: 923–36.
38 Ministry of Industry. Standard Occupational Classification. *Statistics Canada* 1992; 29–43.
39 Davis JA, Smith TW, Hodge RW, Nakao K, Treas J. *Occupational Prestige Ratings from the 1989 General Social Survey*. ICPSR 9593. Inter-university Consortium for Political and Social Research, 1991; 1–131. Available at http://www.icpsr.umich.edu/cgi/archive.prl?num=9593.
40 Hosmer DW, Lemeshow S. *Applied Survival Analysis:*

Regression Modeling of Time to Event. New York: John Wiley & Sons, 1999: 87–270.
41 Allison PD. *Survival Analysis Using the SAS System. A Practical Guide*. Cary, NC: SAS Institute, Inc., 1995; 111–84.
42 Bingham S. Definitions and intakes of dietary fiber. *Am. J. Clin. Nutr.* 1987; **45**: 1226–31.
43 Asp N-G. Dietary fiber – definition, chemistry and analytical determination. *Mol. Aspects Med.* 1987; **9**: 17–29.
44 Moisan J, Meyer F, Gingras S. Diet and age at menarche. *Cancer Causes Control* 1990; **1**: 149–54.
45 De Ridder CM, Thijssen JHH, Bruning PF, Van den Brande JL, Zonderland ML, Erich WBM. Body fat mass, body fat distribution, and pubertal development: a longitudinal study of physical and hormonal sexual maturation of girls. *J. Clin. Endocrinol. Metab.* 1992; **75**: 442–6.
46 Maclure M, Travis LB, Willett W, MacMahon B. A prospective cohort study of nutrient intake and age at menarche. *Am. J. Clin. Nutr.* 1991; **54**: 649–56.
47 Walker AR, Walker BF, Stelma S. Is breast cancer avoidable? Could dietary changes help? *Int. J. Food Sci. Nutr.* 1995; **46**: 373–81.
48 Trichopoulou A, Lagiou P. Worldwide patterns of dietary lipids intake and health implications. *Am. J. Clin. Nutr.* 1997; **66**(Suppl.): 961S–4S.
49 Lipworth L, Martinez ME, Angell J, Hsieh CC, Trichopoulos D. Olive oil and human cancer: an assessment of the evidence. *Prev. Med.* 1997; **26**: 181–90.
50 Massaro M, Carluccio MA, De Caterina R. Direct vascular antiatherogenic effects of oleic acid: a clue to the cardioprotective effects of the Mediterranean diet. *Cardiologia* 1999; **44**: 507–13.
51 Canola Council of Canada. *Canola Oil: Nutritional Properties* [Online]. Available at http://www.canola-council.org/pubs/oilprop.htm. 1999.
52 Wolk A, Bergstrom R, Hunter D, Willett W, Ljung H, Holmberg L, Bergkvist L, Bruce A, Adami HO. A prospective study of association of monounsaturated fat and other types of fat with risk of breast cancer. *Arch. Intern. Med.* 1998; **158**: 41–5.
53 Barrett-Connor E, Friedlander NJ. Dietary fat, calories, and the risk of breast cancer in postmenopausal women: a prospective population-based study. *J. Am. Coll. Nutr.* 1993; **12**: 390–9.
54 Trichopoulou A, Katsouyanni K, Stuver S, Tzala L, Gnardellis C, Rimm E, Trichopoulos D. Consumption of olive oil and specific food groups in relation to breast cancer risk in Greece. *J. Natl. Cancer Inst.* 1995; **87**: 110–6.
55 Martin-Moreno JM, Willett WC, Gorgojo L, Banegas JR, Rodriguez-Artalejo F, Fernandez-Rodriguez JC, Maisonneuve P, Boyle P. Dietary fat, olive oil intake and breast cancer risk. *Int. J. Cancer* 1994; **58**: 774–80.
56 Welsch CW. Relationship between dietary fat and experimental mammary gland tumorigenesis: a review and critique. *Cancer Res.* 1992; **52**(Suppl.): 2040S–8S.
57 Popkin BM, Siega-Riz AM, Haines PS. A comparison of dietary trends among racial and socioeconomic groups in the United States. *N. Engl. J. Med.* 1996; **335**: 716–20.
58 Zacharias L, Rand WM, Wurtman RJ. A prospective study of sexual development and growth in American girls: the statistics of menarche. *Obstet. Gynecol. Survey* 1976; **31**: 325–37.
59 Cooper C, Kuh D, Egger P, Wadsworth M, Barker D. Childhood growth and age at menarche. *Br. J. Obstet. Gynaecol.* 1996; **103**: 814–7.
60 Sitthi-amorn C, Poshyachinda V. Bias. *Lancet* 1993; **342**: 286–8.
61 Dosemeci M, Wacholder S, Lubin JH. Does nondifferential misclassification of exposure always bias a true effect toward the null value? *Am. J. Epidemiol.* 1990; **132**: 746–8.
62 Cummings JH, Hill MJ, Jenkins DJA, Pearson JR, Wiggins HS. Changes in fecal composition and colonic function due to cereal fiber. *Am. J. Clin. Nutr.* 1976; **29**: 1468–73.
63 Kushi LH, Meyer KA, Jacobs DR Jr. Cereals, legumes, and chronic disease risk reduction: evidence from epidemiologic studies. *Am. J. Clin. Nutr.* 1999; **70**(Suppl.): 451S–8S.
64 Williams CL. Importance of dietary fiber in childhood. *J. Am. Diet. Assoc.* 1995; **95**: 1140–6, 9.