Dietary fibre in relation to lung function and respiratory symptoms from childhood to adulthood

Emmanouela Sdona [1,2], Sandra Ekström[1,2], Jenny Hallberg[3,4], Niklas Andersson [1], Niclas Håkansson [1], Alicja Wolk [1], Inger Kull[3,4], Erik Melén [1,3,4] and Anna Bergström[1,2]

[1]Institute of Environmental Medicine, Karolinska Institutet, Stockholm, Sweden. [2]Centre for Occupational and Environmental Medicine, Region Stockholm, Stockholm, Sweden. [3]Department of Clinical Science and Education, Södersjukhuset, Karolinska Institutet, Stockholm, Sweden. [4]Sachs' Children and Youth Hospital, Södersjukhuset, Stockholm, Sweden.

Corresponding author: Emmanouela Sdona (emmanouela.sdona@ki.se)

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In this longitudinal study, there was no consistent association between dietary fibre intake in childhood and lung function or respiratory symptoms up to adulthood. Further research on dietary fibre in relation to respiratory health is needed. https://bit.ly/40Fq1XL

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Abstract

Background Epidemiological studies suggest beneficial associations between dietary fibre intake, lung function and chronic respiratory symptoms in adults. Our aim was to investigate the association between dietary fibre intake in childhood and respiratory health up to adulthood.

Methods The individual fibre intake of 1956 participants from the Swedish population-based birth cohort BAMSE was estimated from 98- and 107-item food frequency questionnaires at ages 8 and 16 years, respectively. At 8, 16 and 24 years, lung function was measured by spirometry. Respiratory symptoms (cough, mucus production, breathing difficulties/wheeze) were assessed by questionnaires, and airway inflammation by exhaled nitric oxide fraction (F_{ENO}) (⩾25 ppb) at 24 years. Longitudinal associations with lung function were analysed by mixed-effects linear regression; associations with respiratory symptoms and airway inflammation were analysed by logistic regression, adjusting for potential confounders.

Results There were no associations between fibre intake at 8 years, as total and from different sources, spirometry measurements and respiratory symptoms at 24 years. Higher fruit fibre intake tended to be inversely associated with airway inflammation at 24 years (OR 0.70, 95% CI 0.48–1.00), which became non-significant after exclusion of participants with food-related allergic symptoms (OR 0.74, 95% CI 0.49–1.10). No associations between fibre intake at 8 and 16 years as an updated lagged exposure and spirometry measurements up to 24 years were observed.

Conclusion In this longitudinal study, we observed no consistent association between dietary fibre intake in childhood and lung function or respiratory symptoms up to adulthood. Further research on dietary fibre in relation to respiratory health across the life course is needed.

Introduction

Chronic respiratory diseases, such as COPD, often originate early in life [1]. Prenatal and early-life environmental and lifestyle factors, including exposure to tobacco smoke and diet, have been implicated in their aetiology, aside from heredity. Epidemiological studies in adults have suggested that a high dietary fibre intake may be associated with a reduced risk of COPD, possibly through modulation of the innate immune response *via* the "gut–liver–lung axis" [2–6]. However, available research has focused on the role of dietary fibre in adulthood.

The health benefits of dietary fibre in relation to cardiometabolic diseases have been well documented [7]. Moreover, a recent systematic review from our group shows that there is suggestive evidence of an association between increased fibre intake and improved lung function in adults [8]. For example, a cross-sectional analysis of 11 897 adults from the Atherosclerosis Risk in Communities study in the USA

indicated that increased total, cereal and fruit fibre are associated with improved spirometry measurements [9]. Moreover, in a prospective study from the same cohort, total fibre intake at baseline was positively associated with forced expiratory volume in 1 s (FEV_1) per forced vital capacity (FVC) 3 years later [10]. Positive associations between fibre intake and lung function have been reported in studies of smokers [11] and patients with asthma [12], as well as among men in a study of healthy adults [13].

Childhood is a critical period for lung development, and exposures in childhood have been shown to influence subsequent respiratory health [14]. To the best of our knowledge, there is no study on the association between dietary fibre intake in childhood and subsequent respiratory health [15]. Thus, the aim of this study was to investigate the association between dietary fibre intake in childhood, as total and from different sources, and lung function and respiratory symptoms up to adulthood.

Methods

Study design

The study was conducted within the prospective birth cohort BAMSE [16], in which 4089 infants aged on average 2 months from predefined areas of Stockholm, Sweden, were recruited (1994–1996). Participants were subsequently followed with repeated questionnaires up to age 24 years. At ages 4, 8, 16 and 24 years, participants were invited to clinical examinations, which included anthropometric measurements, lung function testing and blood sampling using standardised methods. In total, 3064 (75% of original cohort) young adults answered the questionnaire and 2270 (56%) participated in the clinical examination at the 24-year follow-up. The study was approved by the Ethics Committee of Karolinska Institute, Stockholm, Sweden (approval number 2016/1380-31/2), and written informed consent was obtained.

Exposure assessment

Diet at age 8 years was assessed using a food frequency questionnaire (FFQ) including 98 foods and beverages frequently consumed in Sweden, which was answered by a parent (57%) or by a parent together with the child (40%). Participants were asked how often, on average, they had consumed each type of food or beverage during the past 12 months using 10 pre-specified response categories ranging from "never" to "⩾three times/day". At age 16 years, participants were asked to fill in a web-based FFQ including 107 foods and beverages; similar meal-based FFQs have been validated against doubly labelled water and a web-based 7-day weighed food record and showed good validity regarding fibre intake (Spearman's ρ 0.65) [17, 18]. Additionally, fibre intake from a similar FFQ to the one used at the 8-year follow-up has shown good validity in adults (ρ 0.71) [19].

For each FFQ, the frequency of consumption of the food items was converted into mean daily consumption. The individual fibre intake was assessed by multiplying the frequency of consumption of each food item by its fibre content per serving, using composition values obtained from the Swedish Food Composition Database and summarised over foods and beverages [20]. Total energy intake was summarised over the whole diet and calculated using food composition data from the Swedish Food Agency. Fibre intake was adjusted for total energy intake using the residuals method and is presented per mean total energy intake (1900 $kcal \cdot day^{-1}$) [21]. Food items used in the calculation of fibre intake at 8 years have previously been described [22].

Outcome assessment

Lung function was tested according to American Thoracic Society (ATS)/European Respiratory Society (ERS) spirometry criteria at age 8 years using a 2200 Pulmonary Function Laboratory (SensorMedics, Anaheim, CA, USA), at 16 years using a Jaeger MasterScreen-IOS system (Carefusion Technologies, San Diego, CA, USA) and at 24 years using a Vyaire Vyntus system (Vyaire Medical, Mettawa, IL, USA) [23]. The same spirometry test protocol was used at all time points. Z-scores for FEV_1, FVC and FEV_1/FVC were computed for each participant, using the Global Lung Function Initiative (GLI) reference equations [24]. Pre-bronchodilator measurements were available at all follow-ups, and post-bronchodilator 15 min after administration of salbutamol (400 µg) at the 24-year follow-up. Exhaled nitric oxide fraction (F_{ENO}) was measured using a chemiluminescence analyser (EcoMedics Exhalyzer®, Duernten, Switzerland) according to the ATS/ERS guidelines [25]. High F_{ENO} was defined as F_{ENO} ⩾25 parts per billion (ppb) and was considered as a marker of airway inflammation [26].

Respiratory symptoms were assessed by questionnaires at the 24-year follow-up. Chronic bronchitis-like symptoms included cough and mucus production (positive answers to the questions "In the winter, do you usually cough or bring up mucus as soon as you wake up in the morning?", respectively). A positive answer to both was defined as chronic bronchitis [23]. Breathing difficulties/wheeze variable was defined as at least one episode of breathing difficulties or wheeze in the last 12 months.

Statistical analyses

Differences in selected characteristics of study participants were analysed in relation to median fibre intake at 8 and 16 years (separately) by chi-squared and t-test, as appropriate. Correlations between energy-adjusted total fibre intakes at 8 and 16 years, as well as between fibre sources at 8 years, have previously been described [22].

Associations between total fibre intake, as well as fibre sources, at 8 years and pre-bronchodilator lung function at 8, 16 and 24 years were analysed longitudinally by mixed-effects linear regression with a random intercept, an unstructured correlation matrix and restricted maximum likelihood estimation. An interaction term between fibre intake and the time indicator variable was incorporated into the model to estimate age-specific associations and changes in lung function from 8 to 24 years. Associations between fibre intake, airway inflammation and respiratory symptoms at 24 years were analysed using logistic regression. A linearity check was carried out by the Wald test, and no departure from linearity was observed. Associations are presented as per 5 $g \cdot day^{-1}$ increments.

We identified potential confounding factors from previous literature (description in the supplementary material) and then refined our selection by using a directed acyclic graph (DAG) approach [27] (supplementary figure S1). In the multivariable models, we adjusted for sex and total energy intake ($kcal \cdot day^{-1}$) at 8 years, parental education (elementary school, high school, university), parental ethnicity (born in or outside of Sweden), parental history of atopic disease (yes/no), maternal age at delivery <25 years (yes/no), maternal smoking in pregnancy and/or infancy (yes/no), and older siblings (yes/no). Additionally, we further adjusted for smoking at 24 years (yes/no). Fibre sources were mutually adjusted by inclusion in the same model.

Furthermore, we analysed total fibre intake as an updated lagged exposure in the longitudinal models, *i.e.* intake at 8 years was modelled against lung function at 8 and 16 years, and intake at 16 years was modelled against lung function at 24 years.

We stratified analyses by sex, lung function analyses by asthma status and air pollution exposure at 8 years to test potential effect modification. Definition of asthma [28] and assessment of ambient air pollution [29, 30] have previously been described (description in the supplementary material). We also tested possible reverse causation (*i.e.* that the disease would have influenced the exposure) by excluding participants who reported allergic symptoms related to fruits or vegetables, and/or who avoided any of these due to allergic symptoms at age 8 years [22].

Participants were included in the analyses, if information on fibre intake at ages 8 and 16 years with a mean energy intake within ±3 log SD, anthropometric measurements and spirometry at 16 and/or 24 years (n=1956), or information on respiratory symptoms at 24 years (n=1994) were available. A flow chart of the study is shown in supplementary figure S2. All analyses were performed using the statistical software STATA V.16 (StataCorp, College Station, TX, USA).

Results

The study population (n=1956) was generally comparable to the original cohort (n=4089) with regard to distribution of selected characteristics (supplementary table S1), although with a somewhat lower proportion of males (48.3 *versus* 50.5), and a higher proportion of participants with higher socioeconomic status (*e.g.* parent with university education 56.1 *versus* 52.9).

Participants with high fibre intake at age 8 years (⩾ median) were more likely females and had higher socioeconomic status (in terms of parental occupation and maternal age at baseline), and similar distribution of other demographic and lifestyle characteristics compared to those with low intake (table 1). Similar distribution of characteristics was observed in relation to fibre intake at age 16 years (supplementary table S2). Additionally, participants with high fibre intake at 16 years (⩾ median) were more likely to have a high level of physical activity at 16 and 24 years, less likely to smoke at 16 and 24 years, and less likely to be overweight/obese at 24 years compared to those with low fibre intake (supplementary table S2).

Mean pre-bronchodilator FEV_1 z-score and FVC z-score decreased from 8 to 24 years, which is due to fit of GLI references and not due to reduced lung function, as has previously been described [31] (supplementary table S3). Post-bronchodilator spirometry at 24 years is presented in supplementary table S4. At 24 years, prevalence of airway inflammation was 15.2%, chronic bronchitis 5.7%, cough 9.0%, mucus production 15.1% and breathing difficulties/wheeze 24.7% (table 2).

TABLE 1 Distribution of demographic and lifestyle characteristics of study participants by total fibre intake at age 8 years

	Total fibre intake		
	<Median	⩾Median	p-value
Participants n	976	980	
Male sex	506 (51.8)	439 (44.8)	0.002
Maternal age <26 years	84 (8.6)	46 (4.7)	0.001
Parental allergic disease	327 (33.7)	298 (30.7)	0.164
Parent white collar worker	818 (84.9)	857 (88.4)	0.024
Parent with university education	522 (53.5)	574 (58.6)	0.064
Parent born outside of Scandinavia	146 (15.1)	164 (16.8)	0.282
Maternal smoking in pregnancy and/or infancy	128 (13.1)	104 (10.6)	0.085
Breastfeeding ⩾4 months	779 (81.1)	785 (81.6)	0.761
Older siblings	444 (45.5)	462 (47.1)	0.464
8 years			
Overweight or obesity	182 (18.7)	204 (20.8)	0.228
Physical activity >2 times/week	161 (16.5)	141 (14.4)	0.194
Use of multivitamins	411 (42.6)	408 (42.3)	0.906
Asthma	109 (11.2)	106 (10.9)	0.828
Allergy to fruits and vegetables	108 (12.0)	86 (9.6)	0.099
NO_x exposure ⩾median	473 (49.5)	506 (53.6)	0.076
16 years			
Overweight or obesity	170 (18.1)	145 (15.4)	0.120
High physical activity	643 (68.8)	655 (70.2)	0.565
Smoking	120 (12.3)	102 (10.4)	0.186
Allergy to fruits and vegetables	134 (17.4)	153 (20.0)	0.180
24 years			
Overweight or obesity	173 (22.6)	182 (24.2)	0.474
High physical activity	408 (56.5)	433 (59.1)	0.449
Smoking	175 (19.8)	179 (20.0)	0.952
8 years			
Energy intake kcal·day^{-1}, mean±SD	1900±466	1900±445	0.986

Data are presented as n (%) unless indicated otherwise. p-values were calculated by the chi-square test and t-test, as appropriate. Median (IQR) fibre intake 18.0 (4.8) g·day^{-1}. NO_x: oxides of nitrogen.

Associations between fibre intake at 8 years, lung function measurements up to 24 years and respiratory symptoms at 24 years

In mixed-effects linear regression analyses, there was no overall association between total fibre intake at 8 years and spirometry measurements up to 24 years (table 3). No association was observed with cereal, fruit or vegetable fibre sources, while a higher intake of other fibre sources was associated with a decreased mean pre-bronchodilator FVC z-score up to 24 years (−0.20, 95% CI −0.36– −0.04). In age-specific analyses (supplementary table S5), a higher intake of other fibre sources was associated with decreased mean pre-bronchodilator FVC z-score at 16 years (−0.22, 95% CI −0.40– −0.05), while no association was observed at 24 years. Regarding change over time for FEV_1, FVC and FEV_1/FVC per

TABLE 2 Descriptive information on airway inflammation and respiratory symptoms at age 24 years

	Females	Males	Total
F_{ENO} ⩾25 ppb	75 (10.9)	109 (20.6)*	184 (15.2)
Chronic bronchitis	55 (5.4)	53 (5.9)	108 (5.7)
Cough	98 (9.7)	75 (8.3)	173 (9.0)
Mucus production	141 (13.9)	149 (16.5)	290 (15.1)
Breathing difficulties/wheeze	286 (27.5)	205 (21.5)*	491 (24.7)

Data are presented as n (%). F_{ENO} total n=1429 (females n=794, males n=635). Respiratory symptoms total n=1994 (females n=1040, males n=954). F_{ENO}: exhaled nitric oxide fraction. *: p<0.05, statistically significant difference between females and males, calculated by the chi-square test.

TABLE 3 Overall associations between fibre intake at 8 years and pre-bronchodilator lung function up to age 24 years (n=1956)

	Dietary fibre intake at 8 years: β per 5 g·day^{-1} (95% CI)	p-value
Total fibre		
FEV_1 z-score	0.03 (−0.02–0.08)	0.316
FVC z-score	0.03 (−0.02–0.08)	0.316
FEV_1/FVC z-score	0.01 (−0.04–0.06)	0.616
Cereal fibre		
FEV_1 z-score	−0.02 (−0.10–0.07)	0.709
FVC z-score	0.00 (−0.08–0.09)	0.910
FEV_1/FVC z-score	−0.01 (−0.10–0.07)	0.727
Fruit fibre		
FEV_1 z-score	0.03 (−0.05–0.11)	0.421
FVC z-score	0.02 (−0.05–0.10)	0.541
FEV_1/FVC z-score	0.03 (−0.05–0.10)	0.508
Vegetable fibre		
FEV_1 z-score	0.04 (−0.09–0.18)	0.538
FVC z-score	0.07 (−0.06–0.20)	0.299
FEV_1/FVC z-score	−0.04 (−0.17–0.10)	0.606
Other fibre		
FEV_1 z-score	−0.11 (−0.27–0.04)	0.157
FVC z-score	−0.20 (−0.36– −0.04)	0.013
FEV_1/FVC z-score	0.13 (−0.02–0.29)	0.098

Mixed-effects linear regression analyses adjusted for sex, total energy intake at 8 years, parental education, parental ethnicity, parental history of atopic disease, maternal age at delivery <25 years, maternal smoking in pregnancy and/or infancy, and older siblings. FEV_1: forced expiratory volume in 1 s; FVC: forced vital capacity.

fibre intake increments, no difference was observed (data not shown). Further adjustment for smoking at age 24 years had no major influence on the results (data not shown).

In line with the results for lung function, there was no association between total and any fibre source intake at 8 years and respiratory symptoms at 24 years (table 4). A tendency to decreased odds of airway inflammation at 24 years with higher fruit fibre intake at 8 years was observed (OR 0.70, 95% CI 0.48–1.00), but not with total fibre intake (OR 0.96, 95% CI 0.77–1.19). Adjustment for smoking at age 24 years did not influence the results (data not shown).

No interaction between fibre intake and sex, asthma status or air pollution exposure at 8 years regarding lung function was observed (supplementary table S6). A higher total fibre intake at 8 years tended to be associated with decreased odds of airway inflammation at 24 years among participants exposed to oxides of nitrogen (NO_x) ⩾ median (OR 0.78, 95% CI 0.57–1.06), but not among participants exposed to NO_x < median (OR 1.20, 95% CI 0.86–1.67, p-interaction 0.034). No interaction between fibre intake and sex regarding respiratory symptoms was observed (data not shown).

Associations between fibre intake at 8 and 16 years as an updated lagged exposure and spirometry measurements up to 24 years

In mixed-effects linear regression analyses, there were no overall associations between total fibre intake at 8 and 16 years as an updated lagged exposure and spirometry measurements up to 24 years (Table 5), and no difference in change over time for FEV_1, FVC and FEV_1/FVC per fibre intake increments (data not shown).

Sensitivity analyses

After exclusion of participants who reported allergic symptoms related to fruits or vegetables, and/or who avoided any of these due to allergic symptoms at age 8 years (n=194), the association between fruit fibre intake at 8 years and airway inflammation at 24 years became non-significant (OR 0.74, 95% CI 0.49–1.10).

Discussion

In this study of 1956 young adults from the population-based birth cohort BAMSE, we observed no consistent association between total fibre intake in childhood and lung function or respiratory symptoms up to age 24 years.

TABLE 4 Associations between fibre intake at 8 years, airway inflammation and respiratory symptoms at age 24 years (n=1994)

	Dietary fibre intake at 8 years: OR per 5 g·day^{-1} (95% CI)	p-value
Total fibre		
F_{ENO} ⩾25 ppb	0.96 (0.77–1.19)	0.694
Chronic bronchitis	1.25 (0.97–1.62)	0.084
Cough	1.10 (0.89–1.35)	0.397
Mucus production	0.95 (0.80–1.12)	0.539
Breathing difficulties/wheeze	0.93 (0.81–1.07)	0.321
Cereal fibre		
F_{ENO} ⩾25 ppb	1.26 (0.88–1.80)	0.215
Chronic bronchitis	1.44 (0.96–2.18)	0.082
Cough	1.31 (0.93–1.83)	0.121
Mucus production	0.99 (0.75–1.30)	0.920
Breathing difficulties/wheeze	1.00 (0.80–1.26)	0.991
Fruit fibre		
F_{ENO} ⩾25 ppb	0.70 (0.48–1.00)	0.052
Chronic bronchitis	0.98 (0.64–1.48)	0.909
Cough	0.90 (0.64–1.26)	0.534
Mucus production	0.99 (0.77–1.28)	0.953
Breathing difficulties/wheeze	0.84 (0.68–1.04)	0.117
Vegetable fibre		
F_{ENO} ⩾25 ppb	1.47 (0.82–2.65)	0.195
Chronic bronchitis	1.69 (0.88–3.25)	0.116
Cough	1.17 (0.67–2.04)	0.588
Mucus production	0.84 (0.53–1.35)	0.478
Breathing difficulties/wheeze	1.06 (0.73–1.53)	0.763
Other fibre		
F_{ENO} ⩾25 ppb	0.96 (0.49–1.89)	0.908
Chronic bronchitis	1.51 (0.70–3.24)	0.290
Cough	1.47 (0.79–2.73)	0.221
Mucus production	0.96 (0.57–1.62)	0.877
Breathing difficulties/wheeze	1.12 (0.74–1.70)	0.604

Logistic regression analyses adjusted for sex, total energy intake at 8 years, parental education, parental ethnicity, parental history of atopic disease, maternal age at delivery <25 years, maternal smoking in pregnancy and/or infancy, and older siblings. F_{ENO}: exhaled nitric oxide fraction.

Previous studies in adults have indicated that a high dietary fibre intake might protect against reduced lung function and COPD, which is biologically plausible. Fibre is a complex carbohydrate non-digestible component of the diet, which can be found in plant-based foods, such as fruits, vegetables, whole grains, legumes, nuts and seeds [32]. Dietary fibre has been described to contribute to a healthy gut microbiota composition, by promoting the growth of beneficial bacterial species [33]. Additionally, it can have direct and indirect immunomodulatory functions, *via* modulation of the intestinal barrier, as well as production of a wide range of metabolites with immunological properties, such as short chain fatty acids, following

TABLE 5 Longitudinal overall associations between total fibre intake at 8 and 16 years as an updated lagged exposure and lung function from 8 to 24 years (n=1956)

	Total fibre intake at 8 and 16 years: β per 5 g·day^{-1} (95% CI)	p-value
FEV_1 z-score	0.02 (−0.01–0.05)	0.168
FVC z-score	0.02 (0.00–0.05)	0.108
FEV_1/FVC z-score	0.00 (−0.03–0.03)	0.899

Mixed-effects linear regression analyses adjusted for sex, total energy intake, parental education, parental ethnicity, parental history of atopic disease, maternal age at delivery <25 years, maternal smoking in pregnancy and/or infancy, and older siblings. Total fibre intake at 8 years was modelled against lung function at 8 and 16 years, and total fibre intake at 16 years was modelled against lung function at 24 years, and total energy intake was handled similarly. FEV_1: forced expiratory volume in 1 s; FVC: forced vital capacity.

microbial fermentation [34, 35]. Other potential mechanisms include increased antioxidant uptake *via* increased bio-accessibility of antioxidants [36]. The lack of association between fibre intake in childhood, lung function and respiratory symptoms in adulthood in our study may be explained by a true lack of association, or by a limited power to detect associations or by a limited range of exposure. These results are in line with the results of our previous study, in which we observed no consistent association between fibre intake in childhood and asthma up to adulthood [22]. However, we observed a tendency to decreased odds of airway inflammation at 24 years with higher fruit fibre intake, which was somewhat attenuated after exclusion of children with food allergies, indicating that disease-related modification of consumption might have contributed to this [22]. In contrast, we observed a decreased FVC up to 24 years with higher other fibre intake (from potatoes, chips/popcorn, legumes and nuts), which might be a chance finding. Alternatively, other fibre intake might be a proxy of intake of "snacks" and other unhealthy food, although we did not observe a similar tendency to increased respiratory symptoms.

Furthermore, in our study neither sex nor asthma or air pollution significantly modified the associations of total fibre intake with respiratory outcomes. Associations among participants with asthma and smokers have been reported in adult studies [4, 5, 12, 13], which may be explained by the higher oxidative stress in these groups, as well as the continued endogenous production of reactive oxygen species among smokers, even after smoking cessation. Another study found a protective association with lung function in both smokers and nonsmokers [9]. Among nonsmokers, the mechanisms of COPD development may differ from those in smokers and relate more to genetic predisposition or other environmental exposures [37]. In our study, a higher total fibre intake tended to be associated with decreased odds of airway inflammation at 24 years among participants exposed to high NO_x levels, but not among participants exposed to low levels (with significant interaction p-value), which warrants further investigation.

Regarding fibre sources, we observed a tendency to decreased odds of airway inflammation at 24 years with higher fruit, but not with cereal, vegetable or other fibre, intake. Previous studies have found significant associations between cereal and fruit fibre, lung function and COPD [4, 5, 9]. It was hypothesised that cereal and fruit fibre may have physiological properties that are more beneficial to the respiratory system than vegetable fibre, or that this is due to the high uptake of heavy metals, especially cadmium and lead, from vegetables [5]. However, these hypotheses could not be tested in our study.

Major strengths of our study include the population-based prospective longitudinal design and long follow-up time with limited loss to follow-up. Repeated data on lung function and the longitudinal analysis made it possible to study both overall and age-specific associations, as well as change over time. The individual fibre intake was calculated using information from validated FFQs, and our results are in line with estimated fibre intakes in children of corresponding age reported by Swedish studies [38, 39]. Although different FFQ designs were used at 8 and 16 years, these methods are sufficient to rank the participants according to their intake and enabled us to use information from repeated dietary assessments, which may reduce exposure misclassification [40]. We were also able to consider many confounders, using a DAG approach. Despite this, residual or unmeasured confounding cannot be ruled out.

Regarding limitations, selection bias should not be an important problem, as there were small differences in the distribution of characteristics of the study population compared to the total cohort. Respiratory symptoms were assessed through questionnaires, which could lead to some misclassification, though probably non-differential with regards to fibre intake. In addition, objective spirometry and exhaled nitric oxide fraction (F_{ENO}) measurements were used.

According to a recent position paper by the European Academy of Allergy and Clinical Immunology, studies focusing on overall dietary patterns incorporating diverse fibre types and sources may be more relevant than studies on individual fibre in promoting immune health [41]. Current evidence is too limited to recommend any specific types of fibre, so instead a diet rich in vegetables, fruits and whole-grain cereals is recommended [42]. Current recommendations regarding childhood fibre intake are extrapolated from adult data, starting at 13–16 g·day^{-1} for 2-year-olds and increasing until the age of 10 years, when values are comparable with an adult range of 25–30 g·day^{-1} [15]. Of particular importance to understand is the potential timing of associations between dietary fibre intake throughout childhood and immune and respiratory system development up to adulthood. In the meantime, public health messages to increase dietary fibre intake in order to achieve healthier diets from sustainable food systems should be heeded [43].

In conclusion, in this longitudinal study we did not observe a consistent association between dietary fibre intake in childhood and lung function or respiratory symptoms up to young adulthood. Further research on dietary fibre in relation to respiratory health across the life course is needed.

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