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The relationship of dietary patterns with adult lung function and COPD

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ABSTRACT: Previous studies of diet and lung function have focused on associations with individual nutrients and foods, and not dietary patterns.

The relationships between dietary patterns and lung function and spirometrically defined chronic obstructive pulmonary disease (COPD) were investigated in 1,551 males and 1,391 females in Hertfordshire, UK. Dietary information was obtained by food frequency questionnaire and dietary patterns were identified using principal components analysis.

Using regression analysis, after controlling for confounders, a ''prudent'' pattern (high consumption of fruit, vegetables, oily fish and wholemeal cereals) was positively associated with forced expiratory volume in 1 s (FEV_1) (trend p-value <0.001 in males, 0.008 in females) (difference in FEV_1 between top and bottom quintiles of pattern score, 0.18 L (95% CI 0.08–0.28 L) in males, 0.08 L (95% CI 0.00–0.16 L) in females). This pattern was also positively associated with forced vital capacity (FVC) in both sexes. Males with a higher ''prudent'' pattern score had a higher FEV_1/FVC (trend p-value 0.002) and a lower prevalence of COPD (odds ratio comparing top *versus* bottom quintile 0.46, 95% CI 0.26–0.81; trend p-value 0.012). Associations in males were stronger in smokers than nonsmokers (interaction p-value for FEV_1/FVC 0.002).

A ''prudent'' dietary pattern may protect against impaired lung function and COPD, especially in male smokers.

KEYWORDS: Chronic obstructive pulmonary disease, dietary patterns, lung function, principal components analysis, sex, smoking

A reduced forced expiratory volume in 1 s (FEV_1) is a powerful predictor of death from chronic obstructive pulmonary disease (COPD), cardiovascular disease and all causes, even in nonsmokers [1–3]. However, while the strongest risk factor for COPD and accelerated decline in adult lung function is smoking, and smoking cessation leads to a slower decline [4, 5], smoking is thought to explain only a small fraction of the variation in FEV_1 slope; 90% remains unexplained after taking age, height and smoking into account [6]. Hence, identification of other modifiable risk factors for impaired lung function, especially those which might reduce susceptibility to smoking, could have major implications for improving public health and reducing mortality.

Diet is a potentially modifiable risk factor for impaired lung function, and epidemiological studies of the relationship between diet and adult lung function or COPD have tended to focus on associations with intakes of individual nutrients and foods or food groups. Cross-sectional and some prospective data suggest that a higher intake of antioxidants, especially vitamin C, may protect against impaired lung function and COPD [7], although supplementation with antioxidant vitamins does not appear to have a beneficial effect on these outcomes [8].

An alternative approach to trying to investigate highly correlated intakes of individual nutrients and foods, some of which may act synergistically, is to explore associations using dietary patterns, which are typically identified using principal components analysis (PCA). PCA has been used to study relationships between dietary patterns and a number of chronic diseases, including coronary heart disease and colon cancer [9, 10]. Studies of dietary patterns and respiratory outcomes are few to date. While relationships with self-reported chronic bronchitis [11], asthma [12] and COPD [13, 14] have been observed, we are

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not aware of published data on the relationship between dietary patterns and adult lung function. In a UK population, we have investigated relationships between dietary patterns and lung function and COPD.

METHODS

The Hertfordshire Cohort Study

Details about the design of the Hertfordshire Cohort Study (HCS) have been described previously [15]. Briefly, from 1911 to 1948, midwives recorded birth weight and other details on all infants born in the county of Hertfordshire, UK. In 1998, 7,106 males and females born between 1931 and 1939, who were still alive and living in Hertfordshire, were traced using the National Health Service central registry. General practitioners gave permission for us to write to 3,126 (82%) males and 2,973 (91%) females. Of these, 1,684 (54%) males and 1,541 (52%) females agreed to a home interview; 1,579 of these males (94%) and 1,418 of the females (92%) subsequently attended a clinic for spirometry. Complete spirometry and dietary data were available for 1,551 males and 1,391 females. The study had ethical approval from the Bedfordshire and Hertfordshire local research ethics committee and the West Hertfordshire local research ethics committee. All participants gave written informed consent.

Spirometry data

Lung function was measured using a Micro Spirometer (CareFusion UK, Gillingham, UK) in the seated position without noseclips. After at least one practice blow, three FEV_1 and forced vital capacity (FVC) readings were recorded. The highest FEV_1 and FVC values from satisfactory manoeuvres were used in the analyses; these did not necessarily come from the same blow. A bronchodilator was not given before spirometry was performed. For FEV_1, 85.8% of the males and 92.2% of the females had a difference of $\leqslant$0.15 L between their two highest readings; for FVC, the corresponding figures were 80.4% and 88.6%. However, we did not exclude those with differences of $>$0.15 L [16]. The primary outcome of interest was FEV_1. Secondary outcomes included FVC, FEV_1/FVC, and COPD (defined as FEV_1/FVC less than the lower limit of normal, the latter calculated using separate equations for males and females [17]).

Dietary assessment and dietary patterns analysis

Diet was assessed between 1998 and 2004 using a food frequency questionnaire (FFQ) that was based on the European Prospective Investigation of Cancer questionnaire [18]. This was administered by a trained research nurse; consequently, missing information was rare. The FFQ included 129 foods and food groups, and was used to assess an average frequency of consumption of the listed foods over the 3-month period preceding the home interview. Details of the dietary pattern analysis have been reported elsewhere [19]. In brief, the 129 foods listed in the FFQ were put into 51 food groups on the basis of similarity of type of food and nutrient composition. PCA of the reported weekly frequencies of consumption of food groups was used to define patterns of diet [20]. Nutrient supplements were not included in the PCA. A score for each of the dietary patterns identified was calculated for every participant to define an individual's compliance with that pattern. Separate PCA of the males' and females' dietary data identified almost identical patterns of foods, so the data were combined. The first component, which explained the greatest variance in the dietary data, was characterised by high consumption of fruit, vegetables, oily fish and wholemeal cereals, but by low consumption of white bread, added sugar, full-fat dairy products, chips and processed meat. This pattern reflects recommendations for a healthy diet, and we called it a ''prudent'' pattern, in keeping with other studies [13, 14, 21]. The second component was characterised by high consumption of vegetables, processed meat, offal, fish, red meat and puddings but by low consumption of milky drinks, reduced fat spread and breakfast cereals. We called this a ''traditional'' pattern. The foods with the largest factor loadings for these two patterns are shown in table E1 of the online supplementary material. The ''prudent'' and ''traditional'' dietary patterns together explained 13.3% of the variation in consumption of the 51 foods and food groups. Variations in dietary pattern scores indicated the degree of compliance with each pattern, and reflected marked differences in food consumption. For example when comparing the top and bottom fifths of the prudent dietary pattern scores, there were three-fold differences in frequency of consumption of the fruit and salad vegetables among the males and females studied. We also examined a further two components identified by the PCA but they explained less of the variance (3.5% and 3.2%, respectively), and did not define meaningful or interpretable patterns of foods, so we did not consider them further [19].

Statistical analysis

The statistical software package Stata, version 10 (Statacorp LP, College Station, TX, USA) was used to analyse the data. Univariate and multiple linear regression were used to analyse the relationships between dietary pattern scores and lung function outcomes, controlling for age and height and the following potential confounders: smoking status (never, ex, current), pack-years smoked, whether exposed to tobacco smoke in the home, age left education (defined as $\leqslant$14 yrs or $\geqslant$15 yrs), home ownership status (owned/mortgaged, rented or other), number of rooms for household use, number of cars for household use, social class, body fat mass, activity score (0 to 100, derived from frequency of gardening, housework, climbing stairs and carrying loads in a typical week), energy intake, alcohol consumption, dietary supplement use, birth weight, father's social class at subject's birth, use of inhaled or oral steroids and use of paracetamol. Social class was identified on the basis of the subject's own current, or most recent, full-time occupation for males and never-married females, and on the basis of the husband's occupation for ever-married females [22]. Fat mass was calculated by multiplying body weight in kg by body fat percentage. Skinfold thickness was measured at the triceps, biceps, subscapular and suprailiac sites in triplicate. The triplicate values were averaged and then age- and sex-specific DURNIN and WOMERSELY [23] equations were used to estimate body fat percentage. The effect estimates changed little after controlling additionally for waist-hip ratio. Similarly, adding the square of pack-years (in addition to pack-years) to the regression model made little difference to the results for FEV_1, so this variable was not included in the main analyses. In order to examine whether the associations with dietary patterns were modified by smoking, we carried out secondary analyses, stratifying by smoking. We analysed

TABLE 1 Characteristics of the study participants

	Males	Females
Subjects	1551	1391
Age yrs	65.7±2.9	66.6±2.7
Height cm	174.2±6.4	160.9±5.9
Birth weight kg	3.50±0.54	3.34±0.50
Fat mass# kg	23.0±1.4	27.8±1.3
Habitual activity %	61.0±15.3	59.1±15.7
Energy intake# kcal·day^{-1}	2237±1.3	1912±1.3
Smoking status		
Never smoked	507 (32.7)	854 (61.4)
Ex-smoker	806 (52.0)	402 (28.9)
Current smoker	238 (15.3)	134 (9.6)
Pack-years smoked		
Ex-smoker	19 (8–34)	11 (3–23)
Current smoker	36.5 (25–50)	27 (15–40)
Exposed to tobacco smoke in the home	203 (13.5)	163 (11.9)
Alcohol consumption		
Non-drinker	86 (5.6)	273 (19.6)
Exceeded recommended levels¶	336 (21.7)	67 (4.8)
Social class		
I-IIINM	611 (40.6)	583 (41.9)
IIIM-V	896 (59.4)	807 (58.1)
Father's social class		
I-IIINM	231 (15.8)	215 (16.5)
IIIM-V	1,228 (84.2)	1,085 (83.5)
Rooms in household use	5.6±1.6	5.5±1.5
⩾2 cars available for household use	635 (41.0)	353 (25.4)
Owning or mortgaging their home	1,260 (81.2)	1,089 (78.3)
Left full-time education aged ⩾15 yrs	1,249 (80.5)	1,150 (82.7)
Taking dietary supplements	712 (45.9)	824 (59.2)
Taking inhaled steroids	93 (6.0)	107 (7.7)
Taking oral steroids	19 (1.2)	26 (1.9)
Taking paracetamol	105 (6.8)	178 (12.8)
Maximum FEV_1 L	2.84±0.60	1.98±0.41
Maximum FVC L	4.04±0.74	2.71±0.50
FEV_1/FVC ratio	0.70±0.09	0.73±0.08
COPD+	334 (21.6)	187 (13.5)

Data are presented as n, n (%), mean±SD or median (interquartile range), unless otherwise stated. #: geometric mean (SD); ¶: recommended units of alcohol for males, 21 per week; for females, 14 per week; +: chronic obstructive pulmonary disease (COPD) is defined as forced expiratory volume in 1 s (FEV_1)/forced vital capacity (FVC) ratio less than the lower limit of normal.

males and females separately, and tested for interactions of dietary pattern with sex and smoking. Dietary pattern score quintiles were defined using data from males and females combined. We also repeated the analyses using sex-specific quintiles to ensure equal numbers of individuals in each quintile (since the range of dietary pattern scores differed by sex).

RESULTS

The characteristics of the males and females studied are given in table 1. The average age was 66 yrs and participants were almost exclusively white. Males were more likely to have smoked than females, and to smoke more heavily. They were also more likely to drink alcohol, and to exceed recommended limits, than females. Females had a higher fat mass than males, were less likely to come from homes with two or more cars, and were more likely than males to be taking supplements and paracetamol. The prevalence of COPD defined spirometrically was higher in males.

Females had higher mean±SD ''prudent'' diet scores than males (0.72±1.72 *versus* -0.57±2.06), but lower mean ''traditional'' diet scores (-0.33±1.53 *versus* 0.33±1.67). Table E2 in the online supplementary material shows the mean ''prudent'' and ''traditional'' pattern scores by quintile, calculated separately for males and females, and for males and females combined. Predictors of a high ''prudent'' diet score in males and females included being a nonsmoker, of higher socioeconomic status, having a longer education, a lower energy intake, a lower use of paracetamol and a higher use of supplements; a high score was associated with a lower fat mass in females but a higher fat mass in males.

After controlling for all confounders, the ''traditional'' pattern was not associated with any outcome (data not shown). In contrast, the ''prudent'' dietary pattern was positively associated with FEV_1, with evidence of a significant trend ($p<0.001$ in males, $p=0.008$ in females), and no evidence for a threshold effect in either sex. The difference in mean FEV_1 between individuals in the highest and lowest quintiles of pattern score was greater in males (0.18 L) than in females (0.08 L) (table 2), and there was some evidence for effect modification by sex (interaction p-value 0.05). The ''prudent'' pattern was also positively associated with FVC (trend p-value 0.044 in males, 0.007 in females) (table 3). When we analysed the FEV_1/FVC ratio as a continuous outcome, a significant positive association was seen with the ''prudent'' pattern in males (trend p-value 0.002). In females there was no association (table 4). A higher ''prudent'' diet score was associated with a significant reduction in the odds ratio for COPD in males (trend p-value 0.012), with individuals in the top quintile being 54% less likely to have COPD compared with those in the bottom quintile (table 5). In females, there was no association.

When we stratified the effect of a ''prudent'' diet on lung function according to smoking status, there was evidence of effect modification in males but not in females. In males, after controlling for all confounders, the effect estimates were largest in current smokers and intermediate in ex-smokers (interaction p-value comparing current with never smokers, 0.036 for FEV_1 (table 6), 0.002 for FEV_1/FVC (online supplementary table E3), and 0.062 for COPD (online supplementary table E4)). Among never smokers, a ''prudent'' diet was positively associated with FEV_1 in females, but not in males (table 6).

Similar results were obtained when we repeated all the analyses using sex-specific quintiles.

DISCUSSION

We have found that a ''prudent'' dietary pattern was strongly positively associated with lung function, particularly FEV_1, in males and females, and negatively related to COPD in males. The beneficial effects in males were strongest in current smokers. These findings are in keeping with recent prospective studies from the USA, which reported a protective effect of a ''prudent'' dietary pattern on the risk of self-reported COPD,

TABLE 2 Relationship between ''prudent'' dietary pattern and forced expiratory volume in 1 s (FEV_1) in males and females

	Subjects	FEV_1 L	Partially adjusted[#] coefficient	Fully adjusted[¶] coefficient
Males				
Prudent diet score				
1 (lowest fifth)	471	2.69±0.63		
2	360	2.81±0.62	0.06 (-0.01–0.13)	0.08 (0.01–0.16)
3 (middle fifth)	265	2.96±0.54	0.16 (0.07–0.24)	0.14 (0.05–0.22)
4	258	2.90±0.59	0.13 (0.05–0.21)	0.10 (0.01–0.19)
5 (highest fifth)	197	3.01±0.53	0.19 (0.10–0.28)	0.18 (0.08–0.28)
Fifths of prudent diet score as a trend			0.05 (0.03–0.07)	0.04 (0.02–0.06)
			p<0.001	p<0.001
Females				
Prudent diet score				
1 (lowest fifth)	118	1.88±0.42		
2	228	1.91±0.42	0.01 (-0.07–0.09)	0.00 (-0.08–0.08)
3 (middle fifth)	324	1.93±0.42	0.01 (-0.06–0.09)	0.00 (-0.08–0.08)
4	330	1.99±0.42	0.05 (-0.02–0.13)	0.02 (-0.07–0.10)
5 (highest fifth)	391	2.09±0.38	0.13 (0.05–0.021)	0.08 (0.00–0.16)
Fifths of prudent diet score as a trend			0.04 (0.02–0.05)	0.02 (0.01–0.04)
			p<0.001	p=0.008

Data are presented as n, mean±SD or regression coefficient (95% CI), unless otherwise stated. [#]: for age, height, smoking status and pack-years; [¶]: for age, height, smoking status, pack-years, smoke in home, age left education, home ownership status, number of rooms, number of cars, social class, fat mass, activity score, energy intake, alcohol, dietary supplement use, birth weight, father's social class at birth, inhaled or oral steroid use, paracetamol use.

which was also stronger in males [13] than in females [14], although significant interactions with smoking were not found for this outcome [14]. Our ''prudent'' pattern was very similar to that described in the US studies. In contrast, while there were some similarities between our ''traditional'' pattern and a ''Western'' pattern, which was associated with an increased risk of COPD in the US studies, the ''traditional'' pattern differed in terms of its high fish and vegetable content. This

TABLE 3 Relationship between ''prudent'' dietary pattern and forced vital capacity (FVC) in males and females

	Subjects	FVC L	Partially adjusted[#] coefficient	Fully adjusted[¶] coefficient
Males				
Prudent diet score				
1 (lowest fifth)	470	3.92±0.72		
2	359	4.01±0.75	0.04 (-0.05–0.13)	0.05 (-0.04–0.14)
3 (middle fifth)	265	4.13±0.72	0.11 (0.01–0.21)	0.09 (-0.01–0.20)
4	258	4.09±0.73	0.08 (-0.02–0.17)	0.07 (-0.04–0.18)
5 (highest fifth)	196	4.21±0.71	0.16 (0.05–0.26)	0.12 (0.00–0.24)
Fifths of prudent diet score as a trend			0.04 (0.01–0.06)	0.03 (0.00–0.05)
			p=0.003	p=0.044
Females				
Prudent diet score				
1 (lowest fifth)	118	2.61±0.46		
2	227	2.64±0.50	0.00 (-0.10–0.09)	0.03 (-0.07–0.12)
3 (middle fifth)	322	2.64±0.51	-0.12 (-0.10–0.08)	0.01 (-0.08–0.11)
4	330	2.71±0.50	0.06 (-0.03–0.15)	0.05 (-0.04–0.15)
5 (highest fifth)	391	2.83±0.49	0.13 (0.04–0.22)	0.10 (0.01–0.19)
Fifths of prudent diet score as a trend			0.04 (0.02–0.06)	0.03 (0.01–0.04)
			p<0.001	p=0.007

Data are presented as n, mean±SD or regression coefficient (95% CI), unless otherwise stated. [#]: for age, height, smoking status and pack-years; [¶]: for age, height, smoking status, pack-years, smoke in home, age left education, home ownership status, number of rooms, number of cars, social class, fat mass, activity score, energy intake, alcohol, dietary supplement use, birth weight, father's social class at birth, inhaled or oral steroids use, paracetamol use.

TABLE 4 Relationship between ''prudent'' dietary pattern and forced expiratory volume in 1 s (FEV_1)/forced vital capacity (FVC) ratio in males and females

	Subjects	FEV_1/FVC ratio	Partially adjusted[#] coefficient	Fully adjusted[¶] coefficient
Males				
Prudent diet score				
1 (lowest fifth)	470	0.685±0.103		
2	359	0.701±0.091	0.007 (-0.005–0.019)	0.012 (0.000–0.024)
3 (middle fifth)	265	0.719±0.071	0.022 (0.009–0.035)	0.020 (0.007–0.034)
4	258	0.710±0.084	0.018 (0.005–0.031)	0.013 (-0.000–0.027)
5 (highest fifth)	196	0.716±0.068	0.019 (0.005–0.034)	0.025 (0.010–0.040)
Fifths of prudent diet score as a trend			0.005 (0.002–0.009) p=0.001	0.005 (0.002–0.009) p=0.002
Females				
Prudent diet score				
1 (lowest fifth)	118	0.716±0.083		
2	227	0.724±0.083	0.006 (-0.011–0.023)	-0.003 (-0.020–0.014)
3 (middle fifth)	322	0.731±0.078	0.009 (-0.008–0.025)	-0.003 (-0.020–0.014)
4	330	0.731±0.084	0.004 (-0.013–0.020)	-0.008 (-0.025–0.009)
5 (highest fifth)	391	0.742±0.070	0.016 (0.000–0.033)	0.005 (-0.012–0.022)
Fifths of prudent diet score as a trend			0.003 (-0.000–0.006) p=0.055	0.001 (-0.002–0.005) p=0.417

Data are presented as n, mean±SD or regression coefficient (95% CI), unless otherwise stated. [#]: for age, height, smoking status and pack-years; [¶]: for age, height, smoking status, pack-years, smoke in home, age left education, home ownership status, number of rooms, number of cars, social class, fat mass, activity score, energy intake, alcohol, dietary supplement use, birth weight, father's social class at birth, inhaled or oral steroids use, paracetamol use.

TABLE 5 Relationship between ''prudent'' dietary pattern and chronic obstructive pulmonary disease (COPD) in males and females

	Subjects	Subjects with COPD	Partially adjusted[#] OR	Fully adjusted[¶] OR
Males				
Prudent diet score				
1 (lowest fifth)	470	144 (30.6)		
2	359	72 (20.1)	0.67 (0.48–0.95)	0.54 (0.36–0.82)
3 (middle fifth)	265	45 (17.0)	0.61 (0.41–0.91)	0.61 (0.38–0.97)
4	258	45 (17.4)	0.57 (0.38–0.84)	0.62 (0.38–1.00)
5 (highest fifth)	196	28 (14.3)	0.49 (0.31–0.78)	0.46 (0.26–0.81)
Fifths of prudent diet score as a trend			0.84 (0.76–0.92) p<0.001	0.86 (0.76–0.97) p=0.012
Females				
Prudent diet score				
1 (lowest fifth)	118	24 (20.3)		
2	227	40 (17.6)	0.93 (0.51–1.69)	1.28 (0.64–2.56)
3 (middle fifth)	322	50 (15.5)	0.92 (0.52–1.65)	1.43 (0.72–2.85)
4	330	35 (10.6)	0.70 (0.38–1.30)	1.11 (0.54–2.28)
5 (highest fifth)	391	38 (9.7)	0.62 (0.34–1.13)	0.95 (0.47–1.94)
Fifths of prudent diet score as a trend			0.88 (0.77–1.00) p=0.043	0.95 (0.82–1.09) p=0.445

Data are presented as n, n (%) or OR (95% CI), unless otherwise stated. [#]: for age, height, smoking status and pack-years; [¶]: for age, height, smoking status, pack-years, smoke in home, age left education, home ownership status, number of rooms, number of cars, social class, fat mass, activity score, energy intake, alcohol, dietary supplement use, birth weight, father's social class at birth, inhaled or oral steroids use, paracetamol use.

TABLE 6 Relationship between the ''prudent'' dietary pattern and forced expiratory volume in 1 s (FEV_1) according to smoking status

	Never smoked			Ex-smokers			Current smokers		
	Subjects	FEV_1 L	Regression coefficient#	Subjects	FEV_1 L	Regression coefficient#	Subjects	FEV_1 L	Regression coefficient#
Males									
Fifths of prudent diet score									
1 (lowest fifth)	95	2.95±0.53		238	2.74±0.60		138	2.44±0.64	
2	124	2.85±0.59	-0.02 (-0.16–0.12)	191	2.83±0.62	0.09 (-0.01–0.20)	45	2.61±0.68	0.17 (-0.04–0.38)
3 (middle fifth)	121	3.04±0.55	0.07 (-0.07–0.21)	124	2.92±0.52	0.13 (0.01–0.25)	20	2.72±0.53	0.18 (-0.10–0.46)
4	87	3.04±0.53	0.04 (-0.12–0.20)	150	2.85±0.61	0.12 (0.00–0.24)	21	2.70±0.60	0.07 (-0.24–0.37)
5 (highest fifth)	80	3.09±0.59	0.01 (-0.16–0.18)	103	2.93±0.49	0.17 (0.04–0.31)	14	3.11±0.39	0.46 (0.08–0.83)
Fifths of prudent diet score as a trend			0.01 (-0.03–0.05) p=0.574			0.04 (0.01–0.07) p=0.010			0.08 (0.00–0.15) p=0.037
Females									
Fifths of prudent diet score									
1 (lowest fifth)	55	2.00±0.35		38	1.88±0.45		25	1.62±0.41	
2	118	1.99±0.39	0.03 (-0.08–0.14)	68	1.85±0.46	-0.12 (-0.28–0.05)	42	1.77±0.38	0.13 (-0.09–0.36)
3 (middle fifth)	188	1.99±0.39	0.00 (-0.10–0.11)	103	1.84±0.44	-0.12 (-0.28–0.04)	32	1.92±0.45	0.26 (-0.00–0.52)
4	224	2.03±0.40	0.03 (-0.07–0.14)	89	1.86±0.46	-0.13 (-0.30–0.03)	17	2.02±0.36	0.33 (0.05–0.62)
5 (highest fifth)	269	2.11±0.37	0.08 (-0.02–0.19)	104	2.06±0.38	-0.02 (-0.18–0.14)	18	1.97±0.43	0.07 (-0.21–0.35)
Fifths of prudent diet score as a trend			0.02 (0.00–0.04) p=0.033			0.01 (-0.02–0.04) p=0.519			0.03 (-0.03–0.10) p=0.290

Data are presented as n, mean±SD or regression coefficient (95% CI), unless otherwise stated. #: adjusted for age, height, pack-years, smoke in home, age left education, home ownership status, number of rooms, number of cars, social class, fat mass, activity score, energy intake, alcohol, dietary supplement use, birth weight, father's social class at birth, inhaled or oral steroids use, paracetamol use.

may explain why we did not observe a detrimental effect of the ''traditional'' diet on lung function in our study.

A major strength of our study compared to the US studies is that we had objective measures of lung function. This is important for two reasons. First, spirometry is the gold standard means of diagnosing COPD [24] and avoids potential problems of bias that might arise with self-reported disease. Secondly, the implications of our findings, if causal, go beyond COPD, and have relevance for cardiovascular and all-cause mortality, given that lung function is such a strong predictor of these outcomes [1–3]. We did not measure post-bronchodilator lung function, which raises the possibility that a small minority of individuals classified as having ''COPD'' by our spirometric definition may have had asthma. However, if this were the case, this is likely to have led to an underestimation of the effects of a ''prudent'' diet on true COPD, given that prospective studies of older adults have not found a relationship between a ''prudent'' dietary pattern and self-reported asthma [13, 14]. Furthermore, such misclassification of phenotype would seem less likely in males, in whom the effect of the ''prudent'' diet on lung function was modified by smoking, the main risk factor for COPD.

The trend p-values suggest the main results are unlikely to have arisen by chance. Just over half of those invited to take part agreed to home interviews, and over 90% of those performed spirometry. Those who were interviewed were broadly similar in their characteristics to those participating in the nationally representative Health Survey for England [15] and mortality from respiratory disease in males in the Hertfordshire Cohort is similar to that in England and Wales as a whole [25]. We therefore believe that our findings can be extrapolated to the wider English population. While we cannot rule out the possibility that non-response might have biased the associations between diet and respiratory outcomes, for our main findings to be spurious, a higher ''prudent'' dietary pattern score would have to be strongly associated with *worse* lung function in those who did not participate, which seems unlikely. Furthermore, weights at birth and at 1 yr of age, which we have previously shown to be associated with lung function in late adult life and COPD mortality in another Hertfordshire cohort [26], were similar between those that did and did not agree to home interview [15]. We controlled for a large number of potential confounders, including multiple indicators of socioeconomic status, as diet is strongly socially patterned. A strength of our study is that we had reliable information on birth weight, which is unusual in studies of adults of this age. Smoking was also controlled for in some detail. While we cannot rule out the possibility of residual confounding by smoking in males, which might have contributed to the apparently stronger effects in males, this cannot explain the strong effect of the ''prudent'' pattern on FEV_1 which was present among females who had never smoked.

While there is concern that food frequency questionnaires can be prone to measurement error [27], they have been shown to identify similar patterns of diet as other dietary methods, and dietary pattern scores determined using different dietary methods are highly correlated [28–30]. There are conceptual and methodological arguments in favour of using dietary patterns in nutritional epidemiology instead of analysing individual foods and nutrients. Conceptually, people do not eat individual nutrients or foods, but meals, which form a dietary pattern. One methodological advantage of the PCA approach is that it reduces a large number of correlated dietary measurements down to a small number of overall dimensions of diet which are uncorrelated [31]. This also avoids the problem of chance findings arising from multiple statistical comparisons. Another advantage may be that dietary patterns analysis takes account of interactions between nutrients, thus allowing consideration of the effect of the whole diet [32–34].

A limitation of our study is that it was cross-sectional, which limits our ability to infer a causal relationship between diet and lung function and COPD. However, ''reverse causation'' does not seem a likely explanation for the main findings as we cannot see why individuals developing worse lung function and COPD would choose to eat a less healthy diet. Furthermore, the significant trends in the associations, suggestive of a ''dose–response'' effect, the magnitude of the effect and interaction with smoking in males, and the consistency of our findings for COPD with those of recent prospective studies [13, 14], would support a causal interpretation.

The high antioxidant content of fruit and wholemeal bread may underlie the beneficial effect of a ''prudent'' diet on lung function and COPD. Citrus and hard fruits contain high levels of vitamin C and flavonoids, respectively, and whole grains are rich in phenolic acids, flavonoids, phytic acid, avenanthramides, vitamin E and selenium [35]. Observational studies have reported beneficial effects of a higher intake of fruit on adult lung function [36–40], incident COPD risk [41] and COPD mortality [42], and of a higher intake of whole grains on lung function [36] and lower mortality from chronic respiratory disease [43]. Many studies have found a positive association between vitamin C intake and adult lung function [44–49] and there is some evidence in support of flavonoids [50]. The lack of benefit of vitamin supplementation on lung function and hospital admissions for COPD [8] may indicate that observational associations with vitamin C intake were confounded [51], either by other nutrients or by non-dietary lifestyle factors. An alternative explanation is that antioxidant interventions need to be in the form of dietary change, rather than supplementation with individual nutrient pills, in order to be effective. The modification of the ''prudent'' diet effect by smoking in males lends support to an antioxidant mechanism for our findings. Smokers have higher levels of oxidative stress and hence would be expected to benefit the most from a higher dietary intake of antioxidants. We suggest that higher levels of smoking and oxidative stress in males might explain the stronger effect of a ''prudent'' diet in males than in females overall, and the lack of interaction with smoking in females. The high oily fish intake in a ''prudent'' diet may also be responsible for its beneficial effects. While cross-sectional data are contradictory on the effects of fish [52], including fatty fish [40], on lung function, two studies have suggested that a high intake of fish and n-3 fatty acids may particularly protect smokers against impaired lung function and COPD [53, 54]. This is in keeping with the interaction between the ''prudent'' diet and smoking seen in men.

In conclusion, this study suggests that a ''prudent'' dietary pattern may protect against impairment of adult lung function and COPD, especially in male smokers. The ''prudent'' dietary pattern may also reduce cardiovascular and total mortality [55], and this could partly explain why low lung volumes are such strong predictors of these outcomes [1–3]. Appropriate trials are needed to determine whether prevention of COPD and cardiovascular disease in male smokers could be achieved through adoption of a ''prudent'' diet, although such an intervention would be challenging. If the link is causal, the benefits for public health could potentially be substantial.

SUPPORT STATEMENT

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STATEMENT OF INTEREST

None declared.

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