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Does diet modify the effects of air pollution on lung function? A large cross-sectional study

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ABSTRACT

Background: Air pollution and diet both affect lung health and may interact. We investigated whether a healthy diet may modify associations between air pollution and lung function in adults.
Methods: Modelled annual-average concentrations of nitrogen dioxide (NO_2) and particulate matter with aerodynamic diameters ≤10 μm (PM_{10}) and ≤2.5 μm ($PM_{2.5}$) were linked to residential address points of 260,982 individuals in the UK Biobank cohort. Averaged air pollution concentrations in the year of spirometry and two years prior to spirometry measurements were used. The healthy diet score (HDS) was calculated based on dietary data collected at baseline. Effect modifications by HDS and individual food components (fruit and vegetables) on the associations of air pollution and lung function were investigated.
Results: Participants in the highest HDS group had higher forced expiratory volume in 1-s(FEV_1) and forced vital capacity(FVC) than those in the lowest group in both males and females. Interactions between HDS and air pollution were not seen. Suggestive evidence of total fruit intake effect modification on $PM_{2.5}$-FEV_1 was observed in females. Exposure to $PM_{2.5}$ per 5 $\mu g/m^3$ increment was associated with reduced FEV_1 in the low of −14.4 mL (95%CI: −26.8, −2.2) but not in medium and high fruit intake groups (+2.9 mL(95%CI: −13.8,19.7) and +9.7 mL(95%CI: −4.1,23.6), respectively). A similar pattern was observed for FVC.
Conclusions: We found suggestive evidence that higher consumption of fruit may partially reduce the adverse effects of air pollution on lung function in females. These findings merit investigation to see if they replicate in other cohorts.

1. Introduction

Evidence from population-based studies suggests that air pollutants such as particulate matter with aerodynamic diameters ≤2.5 μm ($PM_{2.5}$) and nitrogen dioxide (NO_2) are negatively associated with lung function (Adam et al., 2015; Rice et al., 2015; Lin et al., 2018; Doiron et al., 2019). Further, it has been estimated that the adverse effects of air pollution on lung function may mediate 10%-30% of air pollution associations with mortality (Guyatt et al., 2024), highlighting the importance of identifying potential strategies to reduce the respiratory burden of air pollution.

Diet is a modifiable factor that could confer respiratory health

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through its effects on oxidative stress and inflammation. A number of studies reported that a healthy dietary pattern characterised by high fruit, vegetable, and cereal intake is associated with better lung function (Strachan et al., 1991; Tabak et al., 1999; Okubo et al., 2014; Garcia-Larsen et al., 2015; Wood, 2021; Kelly et al., 2003) (Strachan et al., 1991; Tabak et al., 1999; Okubo et al., 2014; Garcia-Larsen et al., 2015; Wood, 2021; Kelly et al., 2003), likely due to the antioxidant properties found in natural products such as fruit (Arias et al., 2022) and vegetables (Deng et al., 2013). Positive associations have also been found in relation to flavanones and flavonoids, chemical components in fruit and vegetables (Garcia-Larsen et al., 2018; Bondonno et al., 2022, 2024). Higher dietary antioxidant intake is theorised to reduce oxidative damage to lung tissue by scavenging reactive oxygen species (Mudway et al., 2020) and may therefore blunt the effects of air pollution on respiratory health. Recent evidence from asthma populations suggests the vitamin A and D levels were positively associated with lung function, supporting the potential relevance of antioxidant-related micronutrients to respiratory health(Sharma et al., 2026). On the contrary, dietary patterns characterised by high processed meat intake have been reported to be associated with reduced lung function (Okubo et al., 2014; Alessi et al., 2022) and may amplify the negative effects of air pollution by elevating oxidative stress and inflammation, thereby increasing susceptibility to air pollution exposure.

A randomised controlled trial using broccoli sprout beverages, which is rich in compounds that induce antioxidant and detoxification enzymes, reported significantly higher detoxification biomarkers in urine (Egner et al., 2014; Chen et al., 2019), suggesting that dietary antioxidants may partially attenuate oxidative stress induced by pollutants. Nutrition has been identified as an intervention to mitigate the detrimental effects of air pollution (Whyand et al., 2018). To date, evidence on whether diet can modify associations between air pollution and lung function in a large cohort is still limited (Whyand et al., 2018), with studies investigating the dietary effect modification in relation to respiratory symptoms (Govindaraju et al., 2024), chronic obstructive pulmonary disease (COPD) (Ye et al., 2025) and cardiovascular disease mortality risk (Wang et al., 2022).

We have previously published on adverse associations of air pollution with lung function in the UK Biobank (Doiron et al., 2019). The present study aims to examine whether this effect is modified by dietary patterns and food groups. As plant-based foods contain the highest levels of antioxidants (Carlsen et al., 2010), we concentrated analyses of food groups on fruit and vegetable intake. The secondary aim is to investigate whether household income and smoking modify the associations of diet with lung function as there is evidence of stronger negative effects of air pollution observed in groups with lower household income (Doiron et al., 2019) and smoking, which is a well-established respiratory risk factor that promotes oxidative stress(Kluchová and Tkáčová, 2006).

2. Methods

2.1. Study population

The UK Biobank is a large prospective cohort study of approximately 500,000 UK residents. The recruitment took place between 2006 and 2010 (at ages 40-69 years) across 22 assessment centres in England, Wales and Scotland (Allen et al., 2024). During the assessment centre visit, biological samples and physical measurements such as height and spirometry were taken. In addition, participants completed touchscreen questionnaires comprised of general questions and lifestyle-related questions such as dietary choices (Supplementary Table 1).

Participants who performed less than two spirometry manoeuvres were excluded. After the quality control, 349,686 participants had satisfactory spirometry measures. Participants answered 'prefer not to answer' or 'do not know' or reported food intake over the percentile-trimming (1% and 99%) were excluded from the analyses. Of these, 335,389 participants had sufficient information for HDS calculation. In total, 260,982 participants had complete dietary information for HDS calculations, lung function outcome, air pollution exposures and covariates for the main analyses. The derivation of the analytical sample is shown in Fig. 1. Participants with respiratory conditions (COPD, asthma and lung cancer) were left in the main analytic sample to allow broader representation of study populations.

2.2. Lung function measurements

Pre-bronchodilation lung function including forced expiratory volume in 1 s (FEV_1) and forced vital capacity (FVC), was assessed at baseline using a Vitalograph Pneumotrac 6800 spirometer using a standardised protocol under the supervision of trained nurses. The highest spirometry values from acceptable manoeuvres were used in this study, based on previously described acceptability and reproducibility criteria (De Matteis et al., 2016; Shrine et al., 2019).

2.3. Dietary data and healthy diet score calculations

We used data collected at the baseline visit (2006-2010) from a participant-completed touchscreen food frequency questionnaire (FFQ). Bradbury et al. (2018) assessed the validity of the FFQ and reported that it reliably ranked the intake of food components.

We used a dietary pattern analysis approach (Zhao et al., 2021), the Healthy Diet Score (HDS). This identifies commonly consumed food items considered beneficial and/or those that may have harmful effects to health (Liu et al., 2023a; Shang et al., 2023).

Food items were categorised into seven groups to calculate the HDS: total fruits per day (UKB fresh fruit and dried fruit), total vegetables per day (raw/salad vegetable and cooked vegetable), total fish (oily fish and non-oily fish) per week, total processed meat per week, total red meat per week (beef, lamb, and pork intake), total whole grains intake per week (cereal or bread reported as wholemeal or wholegrain bread, bran cereal, oat cereal, and muesli) and total refined grains per week (white bread, brown bread, any other bread, cereal, and other cereals). The intake criteria for each food group and serving definitions are detailed in Supplementary Table 2, with the original questionnaire items provided in Supplementary Table 1.

We adapted the HDS definition based on Liu et al. (2023a) and applied this to the baseline FFQ data. HDS was calculated based on the aforementioned seven food groups. Participants were assigned one point for each of the following favourable dietary components: $\geq$4 servings of fruit/day, $\geq$4 servings of vegetables/day, $\geq$2 servings of fish/week, $\leq$1 serving of processed meat/week, $\leq$1.5 servings of red meat/week, $\geq$3 servings of whole grains/day, and $\leq$1.5 servings of refined grains/day. Points were summed to generate HDS, which were subsequently divided into three groups (low, medium, and high) based on the whole sample distribution and sex-specific distribution-based cut-offs for sex-specific analysis.

2.4. Air pollution exposure assessment

The annual mean concentrations of NO_2, $PM_{2.5}$ and particulate matter with an aerodynamic diameter $\leq$10 μm (PM_{10}) were estimated at 25mx25m spatial resolution using spatiotemporally weighted land use regression models of Europe for 2000-19, which were constructed by the Horizon 2020 EXPANSE project (Shen et al., 2022). The models explained 77%, 62% and 66% of the variation in $PM_{2.5}$, PM_{10} and NO_2 concentrations across Europe, respectively (Shen et al., 2022). The pollution surfaces were linked to participant home address at baseline, geocoded to a 1m resolution.

Three-year average air pollution concentrations were calculated for two years preceding and including the year in which the participant's spirometry measurement (2006-2010) was taken. For example, spirometry measured in 2010, the annual mean concentrations of air

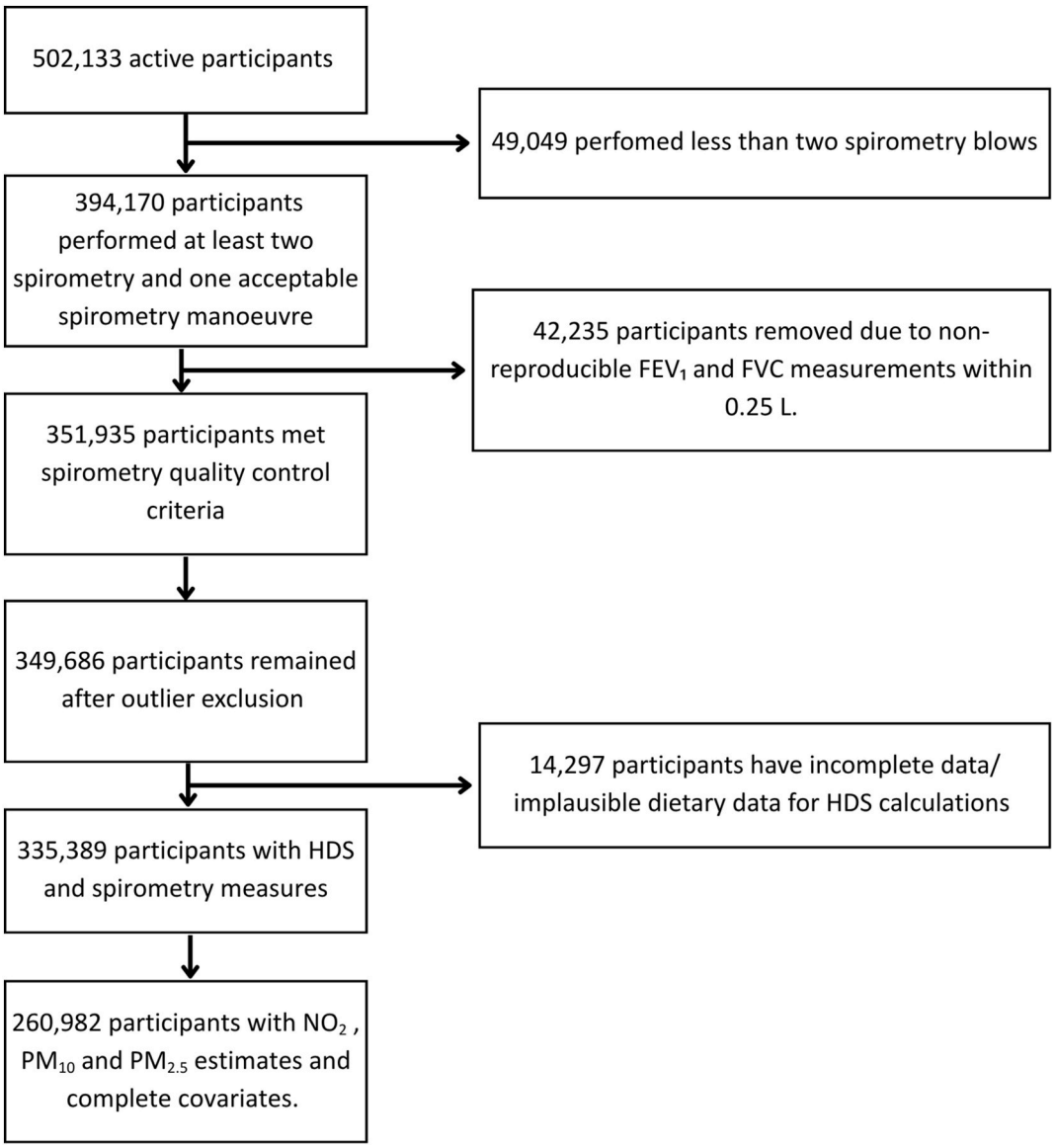

Fig. 1. Participant exclusions and study flowchart.

pollution from year 2008-2010 were averaged.

2.5. Covariates

Confounders and potential effect modifiers were identified a *priori*. Body mass index (BMI) was based on the height and weight measured during the baseline visit and treated as a continuous variable. Educational attainment and household income were treated as categorical variables in their original categories. The Townsend deprivation index was treated as a continuous value. Smoking status was categorised into never, former and current tobacco smokers. Passive smoking was classified into those reporting exposure to second-hand tobacco smoke over an hour per week and those who were exposed less than an hour per week. Physical activity was defined as the number of days that participant reported having vigorous and moderate physical activity for at least 10 min. The assessment centre was adjusted as a proxy for the region. We adjusted for the calendar year of spirometry assessment in analyses involving air pollution for the secular trend of air pollution. The fully adjusted model also included weekly alcohol consumption and self-reported ethnic background (White, Mixed, Asian or Asian British, Black or Black British, and other).

2.6. Statistical analysis

Descriptive analyses were conducted to investigate the baseline characteristics of the study sample by HDS. The HDS was further categorised into low (0–2), medium (3–4), and high (5–7) groups using cut-points based on the distribution of scores in the overall analytical sample for presentation. For subsequent analyses, dietary variables were categorised into low, medium and high intake groups separately using sex-specific cut points based on the observed distribution (Supplementary Table 3).

To assess the relationship between HDS and lung function, we performed linear regression in males and females separately using a complete case analysis, adjusting for age, age-squared, height, BMI (kg/m^2), household income, education level, smoking status (never, former and current), alcohol consumption, physical activity, passive smoking exposure at home, ethnicity background, assessment centre and Townsend deprivation index. The associations were presented in three models to evaluate the effect of confounders on the effect estimates. The lowest HDS group was the reference group in all statistical analyses between HDS groups and lung function (mL). To test for a linear trend, HDS groups were assigned median values.

Effect modification by diet was assessed using linear regression models that included interaction terms between each air pollutant and dietary variable, including HDS, total fruit intake, and total vegetable intake. Evidence for interaction was evaluated using likelihood ratio tests comparing models with and without the relevant interaction term. To aid the interpretation and examine the effect modification of diet on the associations between air pollution ($PM_{2.5}$, PM_{10}, NO_2) and FEV_1 and FVC, stratified linear analyses by HDS groups and fruit/vegetable intake levels were conducted, adjusted for the full set of covariates, including the calendar year. Effect estimates for $PM_{2.5}$ were presented per increment of 5 $\mu g/m^3$, and for PM_{10} and NO_2 were per increment of 10 $\mu g/m^3$. Model adequacy was assessed using residual diagnostics, including checks for normality, homoscedasticity, and influential observations.

To formally assess whether the air pollution-diet interaction differed by sex, a three-way interaction term between sex, air pollution exposure and dietary intake group was included in the combined sample models. The p-value for the three-way interaction was obtained from a joint test of the interaction terms.

2.7. Sensitivity analysis

To evaluate the robustness of the independent associations of HDS and lung function, and the interactions between HDS, total fruit, total vegetables, and air pollutants, we conducted a series of sensitivity analyses. Firstly, we repeated the analyses in a sample with incomplete dietary data for the HDS calculations but with available individual fruit and vegetable intake data. Secondly, participants with asthma, COPD, or prevalent invasive lung cancer at baseline were excluded. COPD was defined using the lower limit of the normal value of pre-bronchodilator FEV_1/FVC ratio, calculated using the Global Lung Function Initiative reference equation. Asthma was defined from self-reported doctor-diagnosed illness. Prevalent invasive lung cancer was defined from cancer registry data (ICD-10 code C34). Thirdly, BMI was excluded as it could be a potential mediator. Fourthly, among ever-smokers with available pack-year data, estimates with and without pack-year adjustment were compared to assess the influence of smoking intensity on diet and lung function association and interaction analyses.

2.8. Subgroup analyses

For our secondary aim, to further explore the effects of smoking and household income on the associations between HDS and lung function, subgroup analyses and interaction tests were conducted. For interpretability, income was categorised into two groups: less than 31,000 GBP and over 31,000 GBP per annum. This is to represent the above- and below-median household income in the UK (Average household income). For smoking, participants were categorised into ever-smokers and never-smokers.

All statistical analyses were performed using R version 4.5.1.

3. Results

Table 1 presents baseline characteristics of the 260,982 participants included in the analysis, grouped into low, medium, and high HDS categories based on the distribution of HDS in the full analytical sample. Around half of these had a medium HDS (50.7%), followed by low HDS (36.2%) and high HDS (13.1%). Participants with higher HDS tended to be older, more often female, more physically active, and less likely to be current smokers or exposed to passive smoking for more than 1 h. Higher HDS was also associated with higher daily servings of total fruit and vegetable intake.

Air pollution levels and Townsend deprivation index showed little variation across HDS groups. Mean FEV_1 and FVC were slightly lower in the high HDS group than in the low HDS group in these unadjusted descriptive comparisons.

Table 1
Descriptive characteristics of the study population at baseline. FEV_1: Forced expiratory volume in 1 s; FVC: Forced vital capacity; $PM_{2.5}$: particulate matter ≤2.5 μm in diameter; PM_{10}: particulate matter ≤10 μm in diameter; NO_2: Nitrogen dioxide; BMI: body mass index. Values are presented as mean (SD) for continuous variables and n (%) for categorical variables unless otherwise indicated.

Characteristic	Overall N = 260,982	Low HDS N = 94,467	Medium HDS N = 132,198	High HDS N = 34,317
FEV_1 (mL)	2892 (754)	2985 (773)	2845 (737)	2819 (738)
FVC (mL)	3784 (961)	3906 (977)	3719 (943)	3698 (951)
Sex				
Female	142,859 (55%)	40,907 (43%)	80,105 (61%)	21,847 (64%)
Male	118,123 (45%)	53,560 (57%)	52,093 (39%)	12,470 (36%)
Age (years)	54 (8)	53 (8)	54 (8)	55 (8)
Height (cm)	169 (9)	170 (9)	168 (9)	168 (9)
BMI (kg/m^2)	27.3 (4.7)	27.9 (4.8)	27.0 (4.6)	26.5 (4.5)
Smoking status				
Never	152,842 (59%)	54,560 (58%)	78,253 (59%)	20,029 (58%)
Previous smokers	100,615 (39%)	36,878 (39%)	50,246 (38%)	13,491 (39%)
Current smokers	7525 (2.9%)	3029 (3.2%)	3699 (2.8%)	797 (2.3%)
Education attainment				
College or University degree	33,150 (13%)	10,070 (11%)	17,880 (14%)	5200 (15%)
A levels/AS levels or equivalent	7982 (3.1%)	2645 (2.8%)	4222 (3.2%)	1115 (3.2%)
O levels/GCSEs or equivalent	50,501 (19%)	18,348 (19%)	25,810 (20%)	6343 (18%)
CSEs or equivalent	17,482 (6.7%)	7316 (7.7%)	8397 (6.4%)	1769 (5.2%)
NVQ or HND or HNC or equivalent	37,363 (14%)	15,215 (16%)	17,823 (13%)	4325 (13%)
Other professional qualifications	114,504 (44%)	40,873 (43%)	58,066 (44%)	15,565 (45%)
Household income				
Less than £18,000	49,174 (19%)	17,889 (19%)	24,250 (18%)	7035 (21%)
£18,000 to £30,999	64,972 (25%)	23,259 (25%)	32,750 (25%)	8963 (26%)
£31,000 to £51,999	71,559 (27%)	26,353 (28%)	36,094 (27%)	9112 (27%)
£52,000 to £100,000	58,959 (23%)	21,440 (23%)	30,321 (23%)	7198 (21%)
Greater than 100,000	16,318 (6.3%)	5526 (5.8%)	8783 (6.6%)	2009 (5.9%)
Ethnicity background				
White	251,036 (96%)	91,503 (97%)	126,780 (96%)	32,753 (95%)
Mixed	1361 (0.5%)	464 (0.5%)	726 (0.5%)	171 (0.5%)
Other Asians	3552 (1.4%)	929 (1.0%)	2067 (1.6%)	556 (1.6%)
Black	2773 (1.1%)	858 (0.9%)	1477 (1.1%)	438 (1.3%)
Chinese	651 (0.2%)	246 (0.3%)	334 (0.3%)	71 (0.2%)
Others	1609 (0.6%)	467 (0.5%)	814 (0.6%)	328 (1.0%)
Alcohol consumption				
Daily or almost daily	55,655 (21%)	22,076 (23%)	27,496 (21%)	6083 (18%)
Three or four times a week	65,757 (25%)	23,876 (25%)	33,503 (25%)	8378 (24%)
Once or twice a week	68,567 (26%)	24,825 (26%)	34,725 (26%)	9017 (26%)
Once to three times a month	28,769 (11%)	10,003 (11%)	14,736 (11%)	4030 (12%)
Special occasions only	25,858 (9.9%)	8475 (9.0%)	13,257 (10%)	4126 (12%)
Never	16,376 (6.3%)	5212 (5.5%)	8481 (6.4%)	2683 (7.8%)
Physical activity (days)	3.66 (2.27)	3.44 (2.29)	3.68 (2.25)	4.19 (2.22)
Passive smoking				

(continued on next page)

Table 1 (*continued*)

Characteristic	Overall N = 260,982	Low HDS N = 94,467	Medium HDS N = 132,198	High HDS N = 34,317
Exposed to other's smoke less than an hour	247,377 (95%)	88,355 (94%)	126,107 (95%)	32,915 (96%)
Exposed to other's smoke greater than an hour	13,605 (5.2%)	6112 (6.5%)	6091 (4.6%)	1402 (4.1%)
$PM_{2.5}$ (μg/m^3)	12.30 (1.88)	12.25 (1.86)	12.32 (1.89)	12.39 (1.90)
PM_{10} (μg/m^3)	20.99 (2.61)	20.89 (2.55)	21.02 (2.63)	21.11 (2.65)
NO_2 (μg/m^3)	25 (8)	25 (7)	25 (8)	25 (8)
Total fruit (servings/day)	2.60 (1.61)	1.87 (1.17)	2.69 (1.53)	4.24 (1.67)
Total vegetable (servings/day)	2.38 (1.29)	1.93 (0.96)	2.40 (1.22)	3.48 (1.64)
Townsend deprivation index	−1.63 (2.88)	−1.62 (2.89)	−1.65 (2.86)	−1.56 (2.92)

3.1. Associations between HDS and lung function

Based on the fully adjusted model, higher HDS was associated with higher FEV_1 and FVC in both females and males, with evidence of a positive trend across the HDS groups. In males, compared with the low HDS group, the high HDS group had 45.8 mL (95% CI: 38.2 to 53.5) higher FEV_1, and the medium HDS group had higher FEV_1 by 28.0 mL (95% CI: 20.2 to 35.7) (Table 2). A similar pattern was observed for FVC, with the high and medium HDS groups having higher FVC by 54.1 mL (95% CI: 45.2 to 63.0) and 26.7 mL (95% CI: 17.7 to 35.7), respectively (Table 2).

In females, the high HDS group showed higher FEV_1 by 36.5 mL (95% CI: 30.7 to 42.3) and the medium HDS group by 19.5 mL (95% CI: 14.7 to 24.2) compared to the low HDS group. For FVC, the differences were 39.6 mL (95% CI: 32.7 to 46.6) for the high HDS group and 20.5 mL (95% CI: 14.8 to 26.2). A test for linear trend was statistically significant, indicating progressively higher lung function across increasing HDS categories (Table 2).

3.2. Effect modification of air pollution–lung function associations by HDS

There was no evidence of interaction between HDS groups and air pollutants for either males or females in relation to FEV_1 and FVC. Although, in stratified analyses by dietary intake level, adverse associations of lung function with air pollution were generally more apparent in the low HDS group, while estimates in the medium and high groups were weaker or closer to the null (Figs. 2 and 3; Supplementary Table 4).

3.3. Effect modification by total fruit and total vegetable intake

In analyses restricted to participants with complete dietary data for HDS calculation, there was a general pattern of lower FEV_1 and FVC with lower fruit or vegetable intake for each air pollutant (Figs. 2 and 3; Supplementary Tables 5 and 6).

However, there was only evidence for a significant interaction between total fruit intake and $PM_{2.5}$ in females for FEV_1 ($P_{int} = 0.02$) and FVC ($P_{int} = 0.04$). Exposure to $PM_{2.5}$ per 5 μg/m^3 increment was associated with lower FEV_1 by 14.4 mL (95% CI: −26.8 to −2.2) for low fruit intake, but non-significant +2.9 mL (95% CI: −13.8 to 19.7), +9.7 mL (95% CI: −4.1 to 23.6), in medium- and high fruit intake groups. A similar pattern was observed for FVC, with corresponding estimates of −18.7 mL (95% CI: −33.3 to −4.1), 3.7 mL (95% CI: −16.4 to 23.8) and 7.7 mL (95% CI: −9.0 to 24.3), respectively. There was no evidence of interaction between fruit intake and PM_{10} or NO_2 (Figs. 2 and 3; Supplementary Table 5). In the combined sample, the formal three-way interaction between sex, $PM_{2.5}$ and fruit intake was not statistically significant for either FEV_1 or FVC ($P_{int} = 0.35$ and 0.55, respectively).

There was little evidence that vegetable intake statistically significantly modified the associations between air pollution and lung function (Figs. 2 and 3). Most interaction p-values were >0.10, although there was suggestive evidence for PM_{10} among males, where adverse associations with FEV_1 appeared stronger in the low vegetable group than in the medium or high groups (−43.6 vs 2.0 and 3.8 mL; $P_{int} = 0.09$). There was no evidence of an interaction between vegetable intake and PM_{10} on FVC. No clear interaction pattern was observed for vegetable intake among females. There was also no evidence of interaction between vegetable intake and NO_2 in relation to lung function for both sexes. (Figs. 2 and 3; Supplementary Table 6).

3.4. Sensitivity analyses

When the analysis was extended to participants with information on fruit and vegetable consumption but not necessarily complete dietary data, the interactions between fruit and $PM_{2.5}$ remained in females (N = 145,979) (Supplementary Table 7). However, in males (N = 120,743), the interactions between total vegetable intake and PM_{10} in relation to FEV_1 became statistically significant with $P_{int} = 0.04$. We observed a stronger adverse association in the low vegetable intake group: (−47.2 mL, 95% CI: −98.7 to 4.3), and little evidence of an association in the high vegetable intake group (3 mL, 95% CI: −32.2 to 38.2) for FEV_1. A similar pattern was observed for FVC, with a stronger adverse association in the low vegetable intake group (−62.9 mL, 95% CI: −122.7 to −3.0) and little evidence of an association in the high

Table 2

Associations between healthy diet score groups and lung function measures. The low healthy diet score (HDS) group was used as the reference category. Effect estimates represent the lung function (mL) compared to the low HDS groups. Values are presented as regression coefficients with 95% confidence intervals (95% CI). Model 1: unadjusted model. Model 2: adjusted for age, age-squared and height. Model 3: adjusted for age, age-squared, BMI, height, household income, educational attainment, smoking status, passive status, physical activity, alcohol intake, ethnic background, assessment centre and Townsend deprivation index. The p-trend represents the significance of linearity across HDS groups.

Sex	Outcome (mL)	HDS groups	Model 1	P for trend	Model 2	P for trend	Model 3	P for trend
Female (N =142,859)	**FEV_1**	**Low**	ref	0.705	ref	<0.001	ref	<0.001
		Medium	7.0 (0.8, 13.2)		22.9 (18.0, 27.8)		19.5 (14.7, 24.2)	
		High	−5.3 (−12.8, 2.2)		38.8 (32.8, 44.8)		36.5 (30.7, 42.3)	
	FVC	**Low**	ref	0.002	ref	<0.001	ref	<0.001
		Medium	16.0 (8.4, 23.6)		29.5 (23.5, 35.5)		20.5 (14.8, 26.2)	
		High	8.7 (−0.5, 17.9)		49.5 (42.3, 56.8)		39.6 (32.7, 46.6)	
Male (N =118,123)	**FEV_1**	**Low**	ref	<0.001	ref	<0.001	ref	<0.001
		Medium	19.1 (9.3, 29.0)		35.9 (27.9, 43.9)		28.0 (20.2, 35.7)	
		High	34.5 (24.8, 44.1)		59.4 (51.6, 67.3)		45.8 (38.2, 53.5)	
	FVC	**Low**	ref	<0.001	ref	<0.001	ref	<0.001
		Medium	26.2 (14.2, 38.1)		39.0 (29.5, 48.6)		26.7 (17.7, 35.7)	
		High	61.0 (49.4, 72.7)		79.0 (69.7, 88.3)		54.1 (45.2, 63.0)	

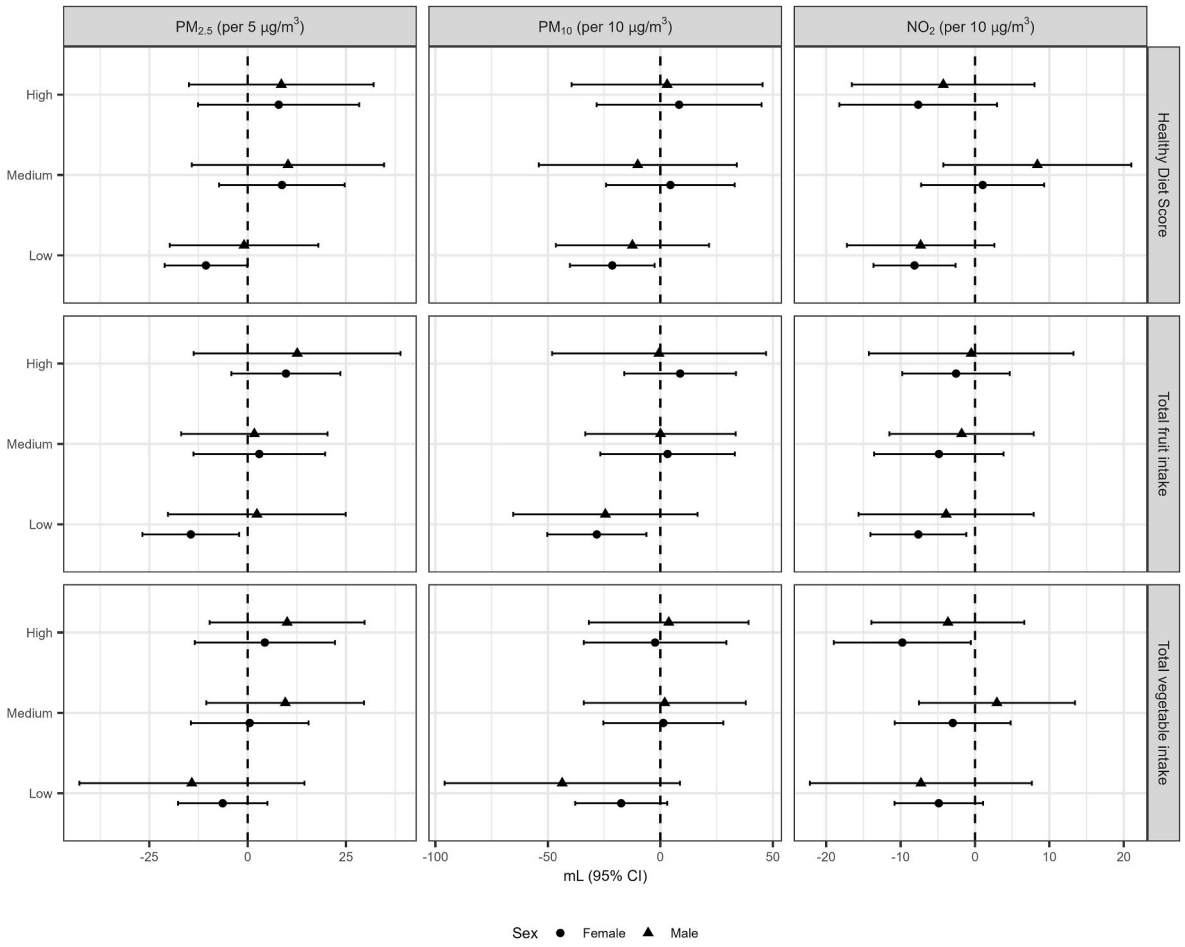

Fig. 2. Associations between air pollution and FEV1 stratified by dietary intake groups and sex. Points show adjusted mean differences in FEV1 (mL) with 95% confidence interval.

vegetable intake group (−7.2 mL, 95% CI: −48.2 to 33.7) (Supplementary Table 8). Those with incomplete dietary data had a higher percentage of current smokers and higher air pollution levels than those with complete dietary data for HDS. The complete HDS group had a higher mean of total fruit and vegetable intake (Supplementary Table 9).

After excluding participants with existing respiratory conditions (~24,000 individuals), the positive association between HDS and lung function remained evident (Supplementary Table 10), significant effect modification of HDS was again not seen (Supplementary Table 11). Evidence of interactions between $PM_{2.5}$ and fruit intake in females remained, although slightly attenuated for FEV_1 ($P_{int} = 0.04$), and the evidence of interactions became weaker for FVC ($P_{int} = 0.06$), but the same pattern was observed: negative estimates in the low fruit intake group and null associations in the medium and high fruit intake groups (Supplementary Table 12). No evidence of significant interactions between total vegetable intake and PM_{10} in men was observed (Supplementary Table 13).

In models excluding BMI, the effect estimates between HDS and lung function were slightly stronger (Supplementary Table 14) and the interactions between fruit intake and $PM_{2.5}$ in females remained in BMI-excluded models for both outcomes (Supplementary Table 15).

The sensitivity analysis regarding smoking intensity adjustment showed that across smokers, higher HDS was consistently associated with higher FEV_1 and FVC in both females and males (Supplementary Table 16). Air pollution-lung function associations stratified by fruit or vegetable intake showed some changes in effect size but not in direction of effect after pack-year adjustment (Supplementary Tables 17–19). For the interaction analyses, additional adjustment for pack-years among smokers modestly changed the magnitude of estimates but did not materially alter the direction or overall pattern of effect modification.

3.5. Subgroup analyses on diet and lung function

In subgroup analyses by smoking status, the magnitude of HDS on lung function was greater among ever-smokers (Supplementary Table 20), with moderate evidence of interaction (P_{int} for $FEV_1 = 0.002$; P_{int} for FVC = 0.01). For subgroup analysis by income, individuals in lower income groups with moderate evidence or borderline significant interaction and a larger magnitude of effect size are shown in Supplementary Table 21. There was moderate evidence of interactions between HDS and income, particularly for FEV_1 ($P_{int} = 0.04$ in females and $P_{int} = 0.005$ in males).

4. Discussion

In this large cross-sectional study, participants on a healthier diet had higher FEV_1 and FVC, as compared to those reporting a less healthy diet. A healthy diet, as assessed by HDS, did not modify the impacts of air pollution on lung function. However, in females, who had higher consumption of fruit, fruit consumption was observed to reduce adverse associations with particulate air pollution.

Despite evidence that HDS is positively associated with lung function in males and females, we did not observe an HDS-related attenuation of the adverse effects of air pollution on lung function. This is in line with a study on COPD incidence (Ye et al., 2025) in the same cohort, using the

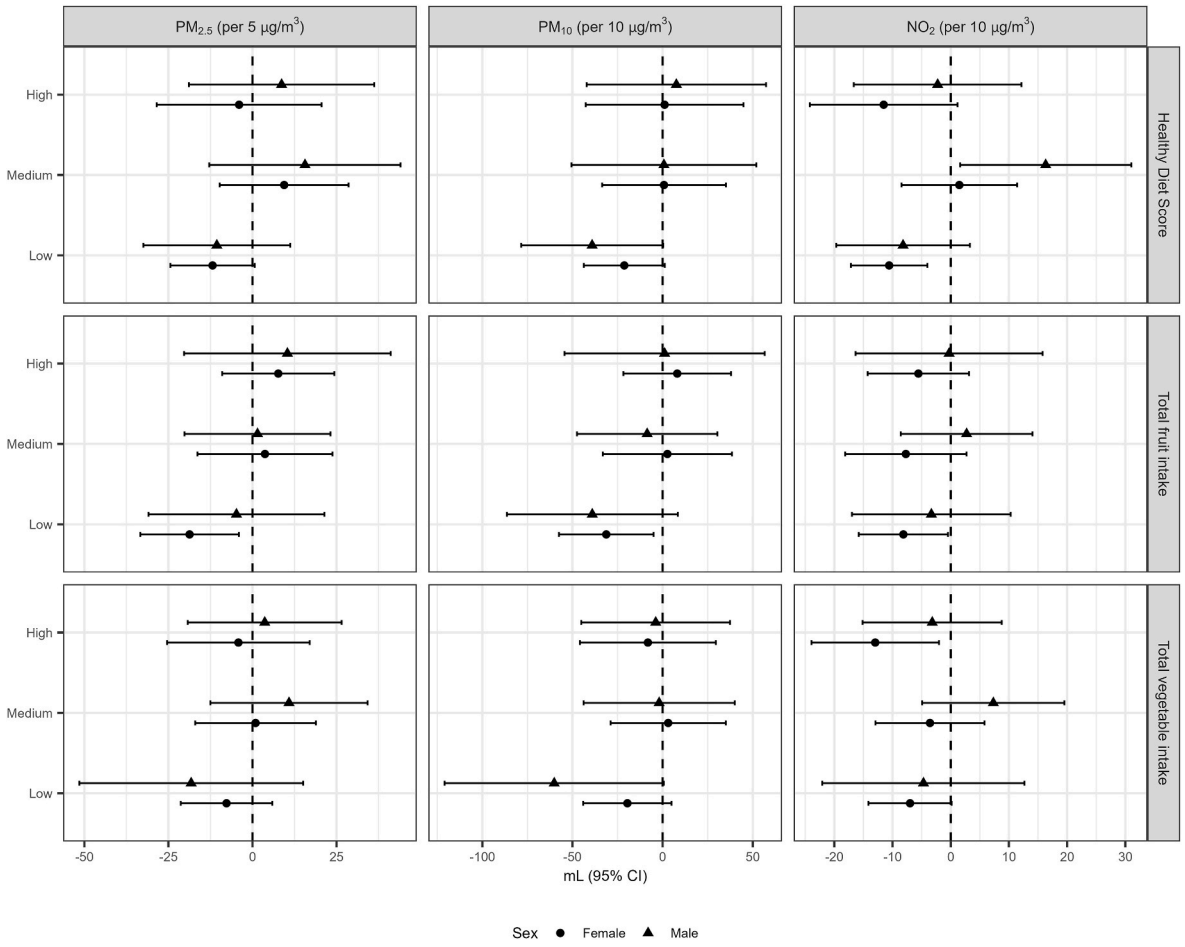

Fig. 3. Associations between air pollution and FVC stratified by dietary intake groups and sex. Points show adjusted mean differences in FVC (mL) with 95% confidence interval.

same dietary groupings. Conversely, another study reported protective effects of higher HDS with respect to air pollution associations with all-cause mortality also using the UK Biobank (Wang et al., 2022). Further studies found protective effects of diet on air pollution and cardiovascular mortality in a large US cohort with greater Mediterranean diet adherence (Lim et al., 2019) and smoke and respiratory symptoms in the Hazelwood coalmine fire study (Govindaraju et al., 2024). These discrepancies may reflect variation of outcome, exposure context and dietary assessment. Alternatively, it may suggest that dietary modification of air pollution and lung function is largely driven by dietary components such as specific antioxidant-rich components, such as fruit and vegetables, rather than by overall diet quality. The HDS used in this study may therefore have limited sensitivity to capture the specific antioxidant or anti-inflammatory pathways most relevant to air pollution-induced oxidative stress in the respiratory system.

We therefore further investigated the potential role of fruit intake and vegetable intake on the associations between air pollution and lung function, hypothesising protective effects due to the high antioxidant properties(Pandey and Rizvi, 2009). We observed evidence of interaction between $PM_{2.5}$ and total fruit intake among females (although not in males with lower fruit intakes). The adverse associations between $PM_{2.5}$ and lung function in females were most apparent in the low fruit intake group and attenuated among those with medium or high fruit intake. The pattern is consistent with that in a previous cross-sectional study of 29,032 adults from six low- and middle-income countries (China, Ghana, India, Mexico, Russia, and South Africa), which found modifying effects of fruit and vegetable intake on the adverse effects of air pollution on lung function in adults at higher levels of air pollution (Mean $PM_{2.5} = 23.6\ \mu g/m^3$; SD = 15.5) compared with our study (Mean $PM_{2.5} = 12.3\ \mu g/m^3$; SD = 1.9). The study reported that each 10 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a smaller reduction of FEV_1 among individuals with high fruit intake ($\beta = -57.7$ mL, 95% CI: −99.5 to −16.5) compared to those with low fruit intake ($\beta = -103.7$ mL, 95% CI: −168.9 to −38.4)(Lin et al., 2018). The study did not conduct sex-stratified analyses, limiting insight into potential sex differences and direct comparison with our female-specific findings. Similar evidence that fruit intake may reduce adverse respiratory associations of air pollution has also been reported for respiratory symptoms (Chen et al., 2019).

Sensitivity analyses generally supported evidence of the fruit–$PM_{2.5}$ interactions for FEV_1 and FVC among females, although the strength of evidence varied across analytical samples. The direction of the estimates was broadly consistent after excluding participants with respiratory conditions, excluding BMI from models and adjusting for pack-years among smokers. More adverse $PM_{2.5}$ associations were observed among females with low fruit intake. In contrast, evidence for the modifying effect of vegetable intake on the PM_{10}-lung function association in males was less consistent across sensitivity analyses, although it was observed when participants with partially complete dietary data were included (an additional ~3,000 females and ~2,500 males). This was less consistent compared to the effect modifications of fruit in females. The lack of effect modification of vegetable intake may partly reflect the lower discriminatory ability of vegetable intake measures or greater measurement error in self-reported vegetable intake compared to fruit intake. A validation study has shown that vegetable intake based on FFQ tends to be less precise than fruit intake, with vegetable intake

more prone to underestimation and misclassification (Kristal et al., 2000). This may partly explain the generally weaker and less consistent associations with lung function (Tabak et al., 1999; Garcia-Larsen et al., 2015).

In terms of plausible mechanisms, fruit is known to have higher antioxidant capacity compared to vegetables (Carlsen et al., 2010); it plays a crucial role in scavenging radical oxidative species induced by exogenous agents such as air pollutants (Romieu et al., 2008). Fruit may also influence gut microbiome composition and diversity, which are involved in regulating oxidative stress levels (Allen et al., 2024), and thus ultimately affect respiratory conditions, including lung function (Liu et al., 2023b). The observation that the interaction was more apparent for $PM_{2.5}$ than PM_{10} may reflect the stronger relevance of fine particles to oxidative stress pathways. It can penetrate deep into the lower respiratory tract which may induce reactive oxygen species and airway inflammation (Xing et al., 2016). As fruit is a source of dietary antioxidants, any potential modifying effect of diet may be more detectable for $PM_{2.5}$.

Although the formal three-way interaction test provided limited evidence that the $PM_{2.5}$-fruit intake interaction differed by sex, we observed the effect modification by fruit in females in sex-stratified analyses. Our stratified findings suggest the potential importance of considering behavioural and biological differences between males and females. A number of possible explanations exist, first, that males are more susceptible to the adverse impacts of air pollution (Doiron et al., 2019; Cai et al., 2020) or are more likely to be in occupations that expose them to high concentrations of air pollution. Thus, the beneficial effects of fruit may not be sufficient to attenuate impacts from ambient air pollution exposure. Second, dietary patterns differed by sex, consistent with the recent Europe-wide survey, which found females generally consumed significantly more fruit and vegetables than males (Stea et al., 2020). Third, sex-related hormonal and metabolic mechanisms variations may also further explain these differences (Liu et al., 2025). Evidence from animal and human studies suggests that males exhibit higher ROS production and lower antioxidant capacity, whereas females exhibit higher antioxidant capacity (Martínez De Toda et al., 2023). Together, higher baseline oxidative stress and greater exposure to oxidant sources, including smoking and air pollution, may partly explain why fruit intake did not clearly modify the associations with air pollution among males. It is possible that a higher level of fruit intake may be required to observe modifying effects in males.

Our findings of a positive relationship between HDS and lung function (when not considering air pollution exposure) are consistent with the current body of evidence. Positive associations between a "prudent" dietary pattern (high consumption of fruit, vegetables, oily fish, and wholemeal cereals) and spirometry measures in both males (N = 1,551) and females (N = 1,391) have been previously reported in an older UK cohort (Shaheen et al., 2010). A stronger association was observed in males, aligning with findings in this study. Another study of ~3000 older individuals in Spain demonstrated a positive association between the Mediterranean dietary pattern and lung function (Gutierrez-Carrasquilla et al., 2019). Notably, we observed a larger magnitude of effects in male smokers, consistent with the prior UK study (Liu et al., 2023a), although residual confounding by smoking intensity cannot be ruled out, as our sensitivity analysis suggests that smoking intensity partly accounts for the associations between diet and lung function. This observation suggests that a healthy diet may be associated with better lung function among males exposed to oxidative stressors such as smoking.

In our UK study population, scores for consumption of healthy food groups were low, particularly for fruit (24.8% of females and 18.3% of males reported ≥4 servings per day) and vegetable intake (13.7% of females and 11.4% of males reported ≥4 servings per day). Further, our subgroup analysis by income revealed that the beneficial impact of a healthier diet on lung function was more pronounced in lower-income groups (Supplementary Table 6), suggesting that promoting diet quality could improve lung health in people living on lower incomes. Studies report higher fruit intake among higher-income groups (Lallukka et al., 2010; Drewnowski and Rehm, 2015). This emphasises the need to improve diet quality in the general population, especially among lower-income groups who are more susceptible to air pollution exposure (Doiron et al., 2019; Hajat et al., 2015). It is important to note that food accessibility and the wider food environment also contribute to food choices, as well as individual preferences (Thomas and Cankurt, 2024). Healthy eating habits should be promoted at policy levels, including reducing barriers to fruit and vegetable access.

The absence of clear negative associations in some stratified models should be interpreted in relation to this study's aim, which was to assess the effect modification of diet rather than to re-quantify the overall effect of air pollution. Our models included adjustment for the assessment centre, which may attenuate pollutant associations by reducing between-area contrasts in exposure. The null estimates within some dietary strata should not be interpreted as evidence that air pollution is not harmful. Air pollution remains an established respiratory risk factor.

This study has several strengths; it is one of the largest studies to investigate the interactions of diet and air pollution exposure on lung function. Even after excluding those with invalid and missing values of dietary data, the UK Biobank provided a large sample size for analyses, which enabled subgroup analyses. In addition, our studies utilised air pollution estimates from a new high-quality model (Shen et al., 2022) at high spatial resolution (25m) assigned to residence geocoded at 1m resolution, allowing for better capture of exposure variability.

Limitations include modelled estimates of air pollution at residence rather than personal exposure, which may lead to some exposure misclassification. We also lacked information on indoor sources of air pollution, although outdoor sources have been found to correlate with indoor air pollution and estimated to contribute ~44% to $PM_{2.5}$ and ~74% to NO_2 concentrations (Evangelopoulos et al., 2020). The use of self-reported diet questionnaires is prone to exposure misclassification and recall bias. We also did not have data on the precise quantity and type of food intake. This was a cross-sectional study, so causal relationships between exposures and lung function cannot be assumed, longitudinal studies are needed to investigate cumulative effects. We acknowledge that residual confounding and reverse causation remain important limitations of this cross-sectional design. To some extent, our sensitivity tests showed that the consistency of HDS-lung function associations after exclusion of participants with respiratory disease reduces the likelihood that reverse causation may explain the findings. We also did not have access to individual level occupation exposure. Occupational exposures to dust, fumes, and other respiratory hazards may differ between men and women and contribute to the observed sex-specific patterns. Although we adjusted for several socioeconomic and lifestyle factors, residual confounding by occupational exposures cannot be excluded. We acknowledge potential for false positive findings due to multiple testing, but were not able to apply formal Bonferroni/FDR adjustments because the interaction tests are highly correlated (shared outcomes, correlated pollutants, and related dietary variables) and because a single objective definition of the multiplicity "family" is not straightforward in this setting (Perneger, 1998). Instead, evidence is presented descriptively by reporting the stratum-specific effect estimates, 95% confidence intervals, and nominal interaction p-values. Findings should be interpreted cautiously; a prospective follow-up of lung function with a more comprehensive dietary assessment or a randomised controlled trial is needed. In addition, we examined individual air pollutants separately to maintain interpretability. Future studies using multi-pollutant or mixture-based approaches are important for public health and may help clarify the combined effects of pollutants. Lastly, the UK Biobank participants are generally more affluent and health-conscious than the general population (Allen et al., 2024), with white ethnicity dominating, which makes our findings less generalisable. Moreover, the dietary patterns in the UK Biobank may have been relatively homogenous and limited the ability of the HDS to

discriminate between distinct levels of diet quality, which may explain the limited evidence of interactions observed. The healthy volunteer bias may dilute the associations between diet and lung function, as well as the ability to detect the effect modification of diet.

5. Conclusion

This study supports previous findings that a healthier diet is positively associated with higher lung function in adults, with increased impacts among lower-income groups and smokers. It further suggests that fruit intake at higher levels may partially reduce the associations between air pollution and lung function in females, highlighting a potential role of antioxidant-rich diets and sex-specific response to air pollution and/or diet. This study warrants further replication in different cohorts.

Support statement

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Ethics statement

This specific project was approved by UK Biobank as part of application 59129. No specific ethical approval was required as the project was covered by the overall approvals for UK Biobank as a whole (all participants provided written consent).

CRediT authorship contribution statement

Pimpika Kaewsri: Formal analysis, Investigation, Methodology, Visualization, Writing – original draft, Writing – review & editing. **Frank Dudbridge:** Methodology, Supervision, Writing – review & editing. **Janet Cade:** Methodology. **Jing Guo:** Methodology, Writing – review & editing. **Xiangpu Gong:** Data curation. **Calvin Jephcote:** Data curation, Writing – review & editing. **Kees de Hoogh:** Data curation, Methodology, Resources, Writing – review & editing. **Roel Vermeulen:** Data curation, Resources. **Marc Chadeau:** Data curation, Resources. **Yutong Samuel Cai:** Methodology, Supervision, Writing – original draft, Writing – review & editing. **Anna L. Hansell:** Conceptualization, Funding acquisition, Methodology, Project administration, Resources, Supervision, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envres.2026.125252.

Data availability

Data will be made available on request.

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