



Dietary antioxidants and forced expiratory volume in 1 s decline: the Health, Aging and Body Composition study

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ABSTRACT: Increased antioxidant defences are hypothesised to decrease age- and smoking-related decline in lung function.

The relationship between dietary antioxidants, smoking and forced expiratory volume in 1 s (FEV₁) was investigated in community-dwelling older adults in the Health, Aging and Body Composition study. 1,443 participants completed a food frequency questionnaire, self-reported smoking history and had measurements taken of FEV₁ at both baseline and after 4 yrs of follow-up. The association of dietary intake of nutrients and foods with antioxidant properties and rate of FEV₁ decline was investigated using hierarchical linear regression models.

In continuing smokers (current smokers at both time-points), higher vitamin C intake and higher intake of fruit and vegetables were associated with an 18 and 24 mL·yr⁻¹ slower rate of FEV₁ decline compared with a lower intake ($p < 0.0001$ and $p = 0.003$, respectively). In quitters (a current smoker at study baseline who had quit during follow-up), higher intake was associated with an attenuated rate of decline for each nutrient studied ($p \leq 0.003$ for all models). In nonsmoking participants, there was little or no association of diet and rate of decline in FEV₁.

The intake of nutrients with antioxidant properties may modulate lung function decline in older adults exposed to cigarette smoke.

KEYWORDS: Ageing, dietary intake, lung function measurements, oxidants/antioxidants, smoking and health

Ageing has been described as the accumulation of oxidative damage that is incompletely repaired by the body's antioxidant defences [1]. This damage is caused by free radicals produced in the body *via* normal metabolic processes and inflammation, and by exogenous free radicals, such as from smoking and noxious gases. In the lungs, ageing is associated with declining lung function, and rate of decline increases with advancing age [2]. In older adults, steeper rates of decline are associated with both an increased risk of chronic obstructive pulmonary disease (COPD) and of all-cause mortality [2, 3]. Among the many harmful effects of cigarette smoking on lung tissue, smoke exposure contributes to oxidative stress and accelerates the "ageing" of the lung, resulting in the greater reductions in lung function observed among people with a history of cigarette smoking [3–5].

The large surface area of the lungs is in constant contact with oxidants from the environment, but antioxidant defences limit the associated damage. When these defences are overwhelmed, oxidative stress can provoke and enhance both the inflammation [6] and antiprotease inactivation [7] associated with COPD, and contribute directly to tissue damage [8]. Enhancing antioxidant defences is hypothesised to reduce the accumulation of oxidative damage, thus potentially slowing the rate of decline in lung function associated with ageing and smoking.

In support of this hypothesis, observational epidemiological studies of dietary antioxidant intake, serum antioxidant concentration and lung outcomes (spirometry at one time-point, rate of decline in lung function and risk of COPD) suggest that lower levels of antioxidant defence

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are associated with worse outcomes [9–14] and higher COPD mortality risk [15]. Although older adults are at the greatest risk of steep rates of decline in forced expiratory volume in 1 s (FEV₁) and may experience the greatest increases in quality of life with prevention, there is no research on the association between dietary antioxidants and FEV₁ in this age group. In addition, most studies of antioxidants and FEV₁ are cross-sectional, despite the expectation that rate of FEV₁ decline is a more informative indicator of mortality and COPD risk [2]. To address the need for longitudinal evidence in older populations, the authors tested the hypothesis that dietary antioxidants modify the association of smoke exposure with the rate of FEV₁ decline in a longitudinal cohort study of older adults.

MATERIALS AND METHODS

Study participants

The Health, Aging and Body Composition Study (Health ABC) is a prospective cohort of 3,075 community-dwelling older adults (aged 70–79 yrs at baseline in 1997; for further details see [16]). The Health ABC study was approved by the Institutional Review Boards of the University of Pittsburgh (Pittsburgh, PA, USA) and the University of Tennessee (Memphis, TN, USA); this study was approved by the Institutional Review Board at Cornell University (Ithaca, NY, USA).

Exclusion criteria included missing dietary or smoking data, or spirometry tests that were missing, unacceptable or not reproducible (early coughs, early termination, high back-extrapolated volume and >200 mL between two best FEV₁ values). Participants with prevalent lung disease (spirometry-defined or diagnosed COPD, or diagnosed asthma) at study baseline were excluded because disease may lead to relevant physiological or behavioural changes.

Study variables

Dietary antioxidant intake was determined at the 1-yr follow-up using a modified Block food frequency questionnaire that was designed to assess intake in the year prior [17]. The intake of nutrients with antioxidant properties was measured by whole food intake (fruit and vegetables) and by micronutrient intake calculated from all dietary sources of the antioxidant (vitamins C and E, and β -carotene).

Spirometry for this cohort was conducted at baseline and after 4 yrs using a horizontal dry rolling seal HF6 Spirometer (Sensor Medics Corporation, Yorba Linda, CA, USA) according to standard guidelines [18]. Smoking exposure was self-reported at both visits and categorised as continuous (smoking at each visit), quit (smoking at baseline but not at 4-yr follow-up), former (history of smoking prior to baseline) and never (smoking <100 cigarettes in lifetime). Smoking pack-yrs and current smoking status were included as covariates.

Data analysis

Hierarchical linear regression models were used to assess the association of antioxidant intake and cigarette smoking with rate of FEV₁ decline. Age, sex, height, race, years of education, family income, study site, pulmonary drug use and total energy intake were included as covariates in all models. Height was included as a repeated measure to address any changes in spirometry secondary to kyphosis. Power terms and categorical variables of continuous antioxidant predictors were

considered; no evidence of nonlinearity was found, thus all models and all statistical tests of the hypothesis used continuous variables for dietary intake. Intake of fruit and vegetables were summed and represented with a single variable (servings per day). To test the interaction between measures of oxidative stress (smoking) and antioxidant status (dietary antioxidant intake, a continuous variable), product terms were computed. Given that interactions were conveyed by a set of variables (for example, the product of each smoking “dummy” variable by antioxidant intake by time), p-values convey the statistical significance of the set of terms. Reported rate of decline estimates are the β coefficients for the elapsed time variable in the model after centering all terms interacting with elapsed time and selecting appropriate smoking reference groups. To illustrate the magnitude of the interaction between smoking and dietary intake, the estimated FEV₁ rate of decline for smokers at high and low antioxidant intake (defined as the 90th and the 10th percentile of the cohort distribution) were calculated.

All analyses were conducted using SAS version 9.1 (SAS Institute, Cary, NC, USA).

RESULTS

Of the 3,075 participants, 859 met exclusion criteria for disease or missing exposure variables (table 1). Of the remaining 2,216 participants, 1,443 had valid spirometry at baseline and follow-up. Inclusion was associated with survivorship and, as expected, studied participants were more likely to be never-smokers or to have smoked less on average over their lifetime.

Although dietary intake variables were modelled as continuous variables, results are presented as the comparison of high (90th percentile) and low (10th percentile) levels for illustrative purposes. In the groups hypothesised to have the highest burden of environmental and endogenous oxidative stress and inflammation (continuing smokers and quitters), a higher intake of nutrients and/or foods with antioxidant properties was consistently associated with a slower rate of decline (table 2); there was little or no association of dietary intake with lung function decline in never- and former smokers. Statistical tests of the interactions of vitamins C and E, β -carotene and fruit/vegetable intake with cigarette smoking were highly statistically significant with p-values of <0.0001, =0.003, =0.003 and =0.003, respectively (full model results provided in online supplementary table 1).

Continuing smokers who consumed diets high in either vitamin C or vitamin E had slower rates of decline in FEV₁ (25 and 29 mL·yr⁻¹) compared with continuing smokers who consumed diets low in these nutrients (43 and 42 mL·yr⁻¹). Continuing smokers who consumed diets high in fruit and vegetables had about half the rate of decline observed in continuing smokers who consumed diets low in fruit and vegetables. The rate of decline in continuing smokers did not differ by intake of β -carotene (table 2).

In smokers who quit, high antioxidant intake was associated with greater attenuation in the rate of decline compared with associations described above for continuing smokers. In quitters with higher intake of nutrients with antioxidant properties, changes in FEV₁ were positive; FEV₁ increased by ~1–2% in high intake groups, whereas it declined by ~2.5% in

TABLE 1 Baseline characteristics of participants included and excluded from analyses, Health, Aging, and Body Composition (Health ABC) study, Memphis, TN, USA and Pittsburgh, PA, USA; 1997–present

	Included participants, by cigarette smoking status				All Health ABC participants	
	Continuing smokers	Quitters	Former smokers	Never-smokers	Included	Excluded [#]
Subjects n	75	27	635	704	1441	1634
Age yrs	72.8±2.8	74.6±3.3	73.5±2.8	73.5±2.8	73.5±2.8	73.8±2.9
Females	39 (52)	10 (37)	229 (36)	451 (64)	731 (51)	853 (52)
Black race	47 (63)	13 (48)	203 (32)	240 (34)	505 (35)	776 (48)
Memphis[†]	30 (40)	11 (41)	278 (44)	332 (47)	652 (45)	896 (55)
Former smokers					640 (44)	764 (47)
Current smokers					97 (7)	221 (14)
Smoking history pack-yrs	44.7±29.0	38.9±34.5	28.3±26.7		30.3±27.7	38.6±31.4
Dietary intake						
Vitamin C mg·day ⁻¹	133±87	149±96	143±72	150±77	146±76	145±80
Vitamin E α-TE·day ⁻¹	11.0±6.0	10.8±6.1	11.2±7.2	11.1±6.8	11.1±6.9	11.0±6.9
β-carotene μg·day ⁻¹	3338±2579	2796±2294	3157±2483	3631±2560	3392±2532	3610±3163
Fruit servings per day	1.6±1.1	1.7±1.2	1.9±1.1	2.2±1.2	2.0±1.2	1.9±1.2
Vegetables servings per day	2.8±2.0	3.1±3.0	3.0±1.9	3.1±1.9	3.0±1.9	2.9±1.9
FEV₁ at baseline[*] mL	2107±48	2198±80	2323±17	2358±16	2297±13	2011±13
Annual rate of decline[§] mL·yr⁻¹	44.3±5.7	31.7±9.4	50.9±2.0	49.4±1.9	49.5±1.3	43.8±2.7

Data are presented as mean±SD or n (%), unless otherwise stated. TE: tocopherol equivalents; FEV₁: forced expiratory volume in 1 s. [#]: number of participants excluded by reason for exclusion (categories not mutually exclusive): prevalent chronic obstructive pulmonary disease (COPD), spirometry-defined (n=357); prevalent COPD, physician-diagnosed (n=228); prevalent asthma, physician-diagnosed (n=140); unacceptable quality on baseline spirometry (n=160); missing baseline spirometry (n=212); unacceptable quality on follow-up spirometry (n=129); missing follow-up spirometry (n=978). [†]: versus Pittsburgh. ^{*}: calculated on those with an acceptable FEV₁ at study baseline, adjusted for age, sex and race. [§]: calculated only on those with an acceptable FEV₁ at both baseline and after 4 yrs of follow-up. Among excluded participants, this calculation is only possible for 354 participants. Values adjusted for age, sex and race.

low intake groups. Thus, high *versus* low intake was associated with a 70-, 96- and 93-mL·yr⁻¹ difference in rate of decline in FEV₁ for vitamins C and E, and β-carotene, respectively. A high intake of fruit and vegetables (*versus* low) was associated with an attenuated rate of decline (attenuated by 41 mL·yr⁻¹; p_{interaction}=0.003) that was similar in magnitude to that observed in continuing smokers.

When dietary vitamins C and E, and β-carotene were included in the same model to account for possible overlap in the effects of these nutrients, a high intake of all three antioxidants (compared with low intake of all) was associated with a 12-mL·yr⁻¹ slower decline in continuing smokers and a 98-mL·yr⁻¹ difference in decline among quitters. The statistical significance of all terms was reduced, and the interaction between vitamin C, smoking status and time had the lowest p-value (p=0.006). Little or no change in the results was observed with the addition of other potentially confounding variables, including time-varying variables for comorbidities (cardiovascular disease, diabetes and hypertension), exercise and time since last smoked.

In analyses of forced vital capacity (FVC), results were less consistent than for FEV₁. There was a statistically significant interaction of vitamin C intake and smoking on rate of decline in FVC (p=0.002), such that current smokers who were high consumers of vitamin C had about half the rate of decline as low consumers. Similar to findings for FEV₁, quitters with high

consumption had positive changes in FVC, whereas quitters with low consumption had rates of decline similar to the overall mean. While the set of vitamin E interactions was statistically significant (p= 0.005), the pattern of effects was less consistent. Thus, the intake of vitamin E had little or no association with rate of decline in FVC in continuing smokers, but quitters with high vitamin E consumption had positive changes in FVC, whereas quitters with low consumption had approximately average rates of decline. Interactions between fruit/vegetable or β-carotene intake and smoking on rate of FVC decline were not statistically significant, although effects were in the same direction as FEV₁ analyses. For the FEV₁/FVC ratio outcome, all effects were in the same direction (less steep declines in smoke-exposed subgroups with higher consumption of nutrients and foods with antioxidant properties), but none of the interaction coefficients were statistically significant.

DISCUSSION

Dietary antioxidant intake is a potentially modifiable predictor of the decline in FEV₁ in this longitudinal study of well-functioning older adults. In Health ABC Study participants, consuming a diet high in antioxidants was associated with an attenuated slope of decline in lung function in current smokers, the population subgroup at greatest risk of lung-related disability and death. In smokers, higher intakes of vitamins C and E, β-carotene, and fruit and vegetables were

TABLE 2 The association of smoking with annual rate of decline in pulmonary function parameters[#] for high (90th percentile) versus low (10th percentile) dietary antioxidant intake in the Health, Aging and Body Composition study, Memphis, TN, USA and Pittsburgh, PA, USA; 1997–present

Nutrient	Smoking status	FEV ₁ decline mL·yr ⁻¹		FVC decline mL·yr ⁻¹		FEV ₁ /FVC decline	
		High intake	Low intake	High intake	Low intake	High intake	Low intake
Vitamin C[†] 236.5 versus 65.2 mg·day⁻¹	Continuous	-25 (-42– -8)	-43 (-58– -29)	-18 (-46– 11)	-40 (-64– -16)	-1.0 (-2.0– -0.1)	-2.4 (-3.1– -1.6)
	Quit ⁺	18 (-13– 49)	-52 (-78– -26)	44 (-7– 95)	-34 (-78– 9)	-2.0 (-3.3– -0.7)	-2.8 (-3.9– -1.6)
	Former	-50 (-57– -44)	-36 (-42– -30)	-46 (-57– -35)	-22 (-32– -12)	-1.3 (-1.6– -1.0)	-1.4 (-1.7– -1.2)
	Never	-42 (-48– -37)	-39 (-45– -34)	-39 (-49– -29)	-34 (-44– -25)	-1.4 (-1.6– -1.1)	-1.3 (-1.5– -1.0)
Vitamin E[‡] 19.5 versus 5.1 α-TE·day⁻¹	Continuous	-29 (-48– -9)	-42 (-57– -26)	-36 (-68– -3)	-29 (-55– -2)	-1.0 (-2.3– 0.3)	-2.4 (-3.3– -1.5)
	Quit ⁺	37 (1– 74)	-59 (-86– -32)	78 (17– 139)	-47 (-92– -1)	-2.0 (-3.4– -0.6)	-2.7 (-3.8– -1.6)
	Former	-45 (-51– -39)	-41 (-46– -36)	-39 (-49– -29)	-28 (-37– -20)	-1.4 (-1.6– -1.1)	-1.4 (-1.6– -1.2)
	Never	-41 (-46– -35)	-41 (-46– -36)	-35 (-45– -25)	-38 (-46– -29)	-1.5 (-1.7– -1.2)	-1.2 (-1.4– -1.0)
β-carotene[‡] 6592 versus 1186 μg·day⁻¹	Continuous	-40 (-59– -20)	-35 (-50– -20)	-37 (-70– -4)	-28 (-54– -3)	-1.7 (-2.8– -0.6)	-2.0 (-2.7– -1.2)
	Quit ⁺	42 (-1– 84)	-51 (-77– -24)	62 (-9– 133)	-28 (-72– 16)	-2.0 (-3.8– -0.2)	-2.2 (-3.2– -1.1)
	Former	-41 (-48– -34)	-43 (-48– -38)	-34 (-45– -22)	-32 (-41– -24)	-1.4 (-1.7– -1.1)	-1.4 (-1.6– -1.2)
	Never	-43 (-49– -37)	-39 (-44– -34)	-38 (-48– -28)	-36 (-44– -27)	-1.3 (-1.6– -1.0)	-1.3 (-1.6– -1.1)
Fruit and vegetables^{###} 8 versus 2 servings·day⁻¹	Continuous	-21 (-41– -1)	-46 (-62– -31)	-12 (-45– 22)	-45 (-71– -18)	-1.6 (-2.7– -0.6)	-2.1 (-2.8– -1.3)
	Quit ⁺	-7 (-33– 19)	-48 (-75– -20)	11 (-33– 54)	-21 (-67– 25)	-2.2 (-3.4– -1.0)	-2.7 (-3.9– -1.6)
	Former	-49 (-56– -42)	-37 (-44– -31)	-39 (-50– -27)	-28 (-39– -17)	-1.4 (-1.7– -1.1)	-1.3 (-1.6– -1.1)
	Never	-42 (-48– -36)	-40 (-46– -33)	-38 (-48– -28)	-35 (-46– -24)	-1.3 (-1.5– -1.0)	-1.3 (-1.6– -1.1)

Data are presented as β (95% confidence interval). FEV₁: forced expiratory volume in 1 s; FVC: forced vital capacity; TE: tocopherol equivalents. [#]: rate of decline estimated from hierarchical mixed models for each nutrient, adjusted for age, height, race, sex, pulmonary drug use, study site, years of education, family income, current cigarettes per day and calorie intake (separate models for each antioxidant, all nutrient variables modelled as continuous variables, but presented as contrasts of high versus low intake to illustrate effect size). [†]: for interaction of vitamin C intake with smoking, p<0.0001 (FEV₁), p=0.002 (FVC) and p=0.1 (FEV₁/FVC). ⁺: quit category is comprised of smokers at baseline who quit during follow-up. [‡]: for interaction of vitamin E intake with smoking, p=0.003 (FEV₁), p=0.005 (FVC) and p=0.2 (FEV₁/FVC). [‡]: for interaction of β-carotene with smoking, p=0.003 (FEV₁), p=0.2 (FVC) and p=0.9 (FEV₁/FVC). ^{###}: for interaction of fruit and vegetable intake with smoking, p=0.003 (FEV₁), p=0.2 (FVC) and p=0.8 (FEV₁/FVC).

associated with meaningful differences in the trajectory of change in FEV₁. In participants who continued to smoke over 4 yrs of follow-up, vitamin C and fruit and vegetable intake had the strongest associations; smokers with high intakes had 18 and 24 mL·yr⁻¹ lower rates of decline in FEV₁ compared with smokers with low intakes. In participants who quit smoking, all dietary antioxidants had strong inverse associations with rate of decline: the rate of decline in FEV₁ was attenuated in quitters with high intakes of vitamin E, β-carotene, vitamin C and fruit compared with quitters with low intake. The association of fruit and vegetables with lung function decline was similar in continuing smokers and quitters, suggesting a broad role for these foods in people who are smoke-exposed. While larger effects were noted for vitamins C and E, and β-carotene in quitters, confidence intervals were wide for this small subgroup.

Prior studies of younger adult populations reported associations between dietary antioxidant intake and rate of decline in FEV₁ similar in magnitude to the Health ABC study findings. Changes over time in the consumption of fresh fruit were associated with rate of decline in FEV₁ in healthy British adults (aged 18–73 yrs): participants with the largest decrease in fruit intake had a steeper rate of decline in FEV₁ (89 mL·yr⁻¹ and 133 mL·yr⁻¹ steeper for males and females) compared with participants with no change in fruit intake [19], and effects were strongest in cigarette smokers. The British study also

reported that average fruit intake (*versus* change in fruit intake) had little or no association with rate of decline, underscoring the importance of appropriately timed measurements of dietary intake. In middle-aged Welshmen (aged 45–59 yrs) followed for 5 yrs, the rate of FEV₁ decline was 10–15 mL·yr⁻¹ slower in males with high apple intake compared with males with low intake; however, no associations were found for vitamins C and E, and β-carotene. Adults in Nottingham, UK (aged 18–70 yrs) consuming diets high in vitamin C (100 mg higher than low vitamin C diets) had an attenuation in the rate of decline in FEV₁ (by about 6 mL·yr⁻¹), but no associations were found for vitamins A and E [12]. In a cohort of heavy-smoking French adults (aged 20–44 yrs), higher levels of serum β-carotene and serum vitamin E were associated with slower rates of decline in FEV₁ [20]. None of the above-mentioned longitudinal studies are in the age range reported herein. Similarly, most of the cross-sectional studies that have investigated this hypothesis are in younger populations, and most reported positive associations between vitamins C and E, β-carotene, fruit and vegetables and lung outcomes [9–15, 19–25], with the strongest effect sizes often identified in cigarette smokers.

In Health ABC never- and former smokers, the intake of nutrients and foods with antioxidant properties had little or no association with the rate of decline in FEV₁. Similarly, when antioxidant terms were included in models without interactions

with smoking status, the main effects were not statistically significant (data not shown). These findings are consistent with the hypothesis that a protective effect of nutrients with antioxidant properties is expected primarily in persons with a high burden of oxidative stress. Thus, the role of dietary antioxidants may be to provide additional functional capacity to reduce damage from exposure to oxidants. The findings suggest a testable hypothesis: nonsmokers may not benefit from increasing the intake of nutrients with antioxidant properties because their overall level of oxidant burden is low enough to be well-managed by endogenous capacity.

The Health ABC study allows a closer look at the rate of decline in lung function associated with smoking status in a cohort of older adults. Of particular interest is the difference in rate of decline in FEV₁ between continuing smokers (44 mL·yr⁻¹) and quitters (31 mL·yr⁻¹), underscoring the significant functional improvements that may be attained with smoking cessation at advanced ages. Attenuating the rate of decline is especially important in smokers because, on average, smokers have lower starting FEV₁ values, and are at greater risk of disability and death.

In the Health ABC study, spirometry was conducted beyond the time-points studied, with additional measurements at 7 and 9 yrs of follow-up. Given that exposure (dietary antioxidants) was measured only once early in the follow-up period, it was important to carefully consider the optimal outcome to investigate the hypothesis. If the balance between antioxidants and oxidants in the lungs is hypothesised to affect the rate of decline in lung function, then concurrent measurements of diet, smoking parameters and lung function are likely to be most informative. Thus, the longitudinal spirometry outcome data were limited to the time period closest to the dietary assessment time-point. When the analysis was extended to include spirometry from later visits (7 and 9 yrs), the magnitudes of the associations were similar, but less statistically significant. In this vulnerable age group, many factors are associated with dietary changes, including new disease diagnoses, onset of functional impairment, changes in living situation, and loss of a spouse [26–28]. Thus, the strongest analytical approach is one that limits consideration of pulmonary function changes to the same time period in which diet was measured.

In any observational study of diet, unaddressed confounding is a concern in interpreting results. Variables representing the following potential confounders of the diet-rate of FEV₁ decline associations were considered in all models: body mass index, cigarettes per day and environmental oxidant exposure (study site, education and family income were used as proxies for environmental exposures). There was little or no change in the reported associations when potential confounding by comorbidities (covariates included prevalent and incident diagnoses of cardiovascular disease, diabetes and hypertension) and exercise/physical activity were considered.

While the hypothesis posits an effect of diet on the oxidant/antioxidant balance, the diet-rate of decline associations may be mediated through other biological pathways, such as muscle strength or inflammation. The finding of similar associations with FVC raises the possibility of other pathways,

although findings for the FVC outcome were less consistent. These data cannot definitively address the biological mechanisms, but support the need for further mechanistic studies.

The use of Global Initiative for Chronic Obstructive Lung Disease (GOLD) categories for defining prevalent COPD has been debated, especially in older populations [29]. When participants were excluded on the basis of lower limit of normal-defined COPD instead of GOLD-defined COPD, neither the size nor the strength of the observed associations was affected.

A strength of this study is the use of data from a large, prospective cohort study of elderly people; this population subgroup stands to benefit from research aimed at understanding how to prevent loss of lung function. The quality control standards for spirometry in the Health ABC study followed best practice, as evidenced by the excellent overall quality of the pulmonary function data. The use of two separate methods to estimate dietary intake of nutrients with antioxidant properties, micronutrients and whole foods, yielded similar results, suggesting that the findings represent true associations of diet and lung function. The consideration of simultaneous models including all nutrients diminishes the possibility that observed associations were primarily driven by a single dietary constituent.

There are several limitations of the study. While the use of antioxidant supplements is expected to contribute to overall antioxidant status, this study did not record the dose and frequency of antioxidant supplement use. Survivor bias is of concern in a study of older adults, *i.e.* nonrandom loss-to-follow-up results in healthier participants at follow-up. Survivor bias is evident when participants contributing data at each measurement are compared: participants with follow-up data were younger, more often female and had smoked less. If participants who did not remain in the study had a greater burden of oxidative stress (given their smoking history and age, this may be a reasonable hypothesis) then, in light of the findings in participants with the highest burden of oxidative stress, even stronger associations than those observed might be expected.

This study of older adults supports the hypothesis that nutrients with antioxidant properties are associated with a slower rate of FEV₁ decline in current and quitting smokers. Whether changing the intake of foods rich in nutrients with antioxidant properties will lead to a slower rate of decline in FEV₁ remains to be demonstrated; the results from the Health ABC cohort support this inference.

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STATEMENT OF INTEREST

None declared.

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