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Association of dietary calcium intake with chronic bronchitis and emphysema

Xuefang Li[1], Zhijun Li[2], Jian Ye[2] and Wu Ye[2*]

Abstract

Objective Chronic bronchitis and emphysema (CBE) are two main types of chronic obstructive pulmonary disease (COPD). We aimed to investigate the relationship between dietary calcium intake and the risk of CBE.

Methods Data were obtained from the National Health and Nutrition Examination Survey (NHANES) 2007–2012. The ratio of forced expiratory volume in 1 s (FEV1) to forced vital capacity (FVC) < 0.7 was used to define airflow obstruction. Multivariate logistic regression was performed to assess the effects of dietary calcium intake on CBE and airflow obstruction. Dietary calcium intake was divided into quartiles, with the lowest quartile set as the reference group. Linear regression models were applied to explore the association between dietary calcium intake and lung function.

Results A total of 10,143 participants were enrolled in the study, including 594 CBE and 9549 non-CBE individuals. The average dietary calcium intake was 908.5 ± 636.1 mg/day in the CBE group and 951.9 ± 599.7 mg/day in the non-CBE group. When using the lowest quartile of dietary calcium intake as a reference, the second, third, and fourth quartiles reduced the risk of CBE by 0.803 [95% confidence interval (CI): 0.802–0.804; $P < 0.001$], 0.659 (95% CI: 0.659–0.660; $P < 0.001$) and 0.644 (95% CI: 0.643–0.644; $P < 0.001$) times, respectively. Increased dietary calcium intake was correlated with reduced risk of airflow obstruction. Dietary calcium intake positively predicts FEV1 ($\beta = 0.225$, $P < 0.001$) and FVC ($\beta = 0.232$, $P < 0.001$).

Conclusion Increased intake of dietary calcium may contribute to higher lung function, a lower risk of CBE and airflow obstruction. Since the cross-sectional design makes it difficult to determine a causal relationship, further research is needed to confirm these findings and explore the underlying mechanisms.

Keywords Chronic bronchitis, Emphysema, Dietary calcium intake, National health and nutrition examination survey, Airflow obstruction, Lung function

*Correspondence:
Wu Ye
yewu55@126.com
[1]Department of Infectious Diseases, Zhejiang Hospital, 1229 Gudun Road, Xihu District, Hangzhou 310013, Zhejiang Province, People's Republic of China
[2]Department of Respiratory Diseases, Zhejiang Hospital, 1229 Gudun Road, Xihu District, Hangzhou 310013, Zhejiang Province, People's Republic of China

Introduction

Chronic bronchitis is defined as chronic productive cough for at least 3 months per year for 2 consecutive years [1]. Emphysema is an anatomical diagnosis defined as permanent dilation, destruction and abnormality of the distal airspaces [2]. Chronic bronchitis and emphysema (CBE) are two main types of chronic obstructive pulmonary disease (COPD), which is currently the third major cause of death worldwide [3–4]. COPD is one of the most common chronic diseases of respiratory system, with a prevalence ranging from 3.6 to 10.1%. Cigarette smoking is a major risk factor of COPD. However, only 15–20% of current or former smokers develop COPD in their lifetime. Therefore, future studies are needed to explore other risk factors involved in the development of COPD [5].

The main characteristic of COPD is irreversible and progressive airflow obstruction. The ratio of forced expiratory volume in 1 s (FEV1) to forced vital capacity (FVC) < 0.7 was used to define airflow obstruction [6]. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) employs airway obstruction for diagnosis of COPD and staging the disease severity. The FEV1/FVC ratio and FEV1 are used to assess the severity of airway obstruction [7–8].

Nutrition plays a critical role in both prevention and treatment of COPD [9–10]. Diets rich in fruits, vegetables, fish and whole grains showed lower risk of newly identified COPD [10]. Calcium is one of the most abundant mineral elements widely related to various physical activities [11]. Adequate dietary calcium intake contributes to skeletal system development and prevention of bone loss [12–13]. Dietary calcium affects many physiologic activities, including muscle function, bone health and growth, kidney function, gluconeogenesis, glycolysis, parathyroid performance, and intestinal function [14].

Insufficient dietary calcium is involved in numerous health conditions such as hypertension, type 2 diabetes, preeclampsia, colon and rectal cancers, osteoporosis and cognitive function [15–16]. Calcium ion modulates airway inflammatory responses [17]. The regulation of intracellular calcium ion in airway smooth muscle is related to several respiratory diseases, including COPD, asthma, and pulmonary fibrosis [18]. However, the association between dietary calcium intake and CBE was not fully understood. The main purpose of our study was to explore the effects of dietary calcium intake on CBE and airflow obstruction using comprehensive data from the National Health and Nutrition Examination Survey (NHANES).

Materials and methods

Data sources and study population

NHANES is conducted every two years by the U.S. Centers for Disease Control and Prevention. NHANES data includes the health and nutritional status of the American population. Individuals participated in the 2007–2008, 2009–2010, and 2011–2012 NHANES survey were integrated into our study. Ethics approval was accepted by National Center for Health Statistics Research Ethics Review Board, and all participants provided informed consent.

A total of 29,139 individuals were identified from NHANES 2007–2012. We excluded missing data, including age, gender, race, education, ratio of family income to poverty, marital status, body measures, dietary calcium, FEV1, FVC and smoking. FEV1 or FVC with the quality of C, D or F was also excluded. Finally, 10,143 participants were included in our study, and the flow chart of the screening process was shown in Fig. 1.

Study variables

We collected the following data of participants, including age, gender (male and female), race (non-Hispanic white, non-Hispanic black, Mexican American, other Hispanic, and other races), marital status (married, widowed, divorced, separated, never married, and living with a partner), education level (under grade 9, grades 9–11, high school graduates, some college/AA degrees, and college graduates), ratio of family income to poverty (≤ 1, 1–2, 2–4, and > 4) and body mass index (BMI) (≤ 18.50, 18.51-25, 25.01-30, and > 30). BMI is calculated by dividing body weight in kilograms by the square of height in meters (kg/m^2). Smoking status was divided into the following two groups: non-smokers (smoked less than 100 cigarettes in a lifetime) and smokers (smoked at least 100 cigarettes in a lifetime). Dietary data were obtained from the 24-hour diet questionnaire in NHANES. The multipass approach method was used to assess precise consumption data of all foods and beverages. NHANES dietary interviewers' procedure manuals contained a comprehensive overview of dietary interview methodologies. We selected 24-hour recall dietary interview data for the first day in the study. Dietary calcium intake is skewed distribution data. Thus, the continuous concentration of dietary calcium intake was divided into quartiles, with the lowest quartile set as the reference group. Participants who are informed by a doctor or other healthcare provider (HCP) to have emphysema and/or chronic bronchitis are considered to have CBE. FEV1 and FVC were obtained from pre-bronchodilator spirometry. The FEV1/FVC ratio < 0.7 was defined as airflow obstruction. To ensure the accuracy of the results, we only included FEV1 and FVC data with quality levels A or B.

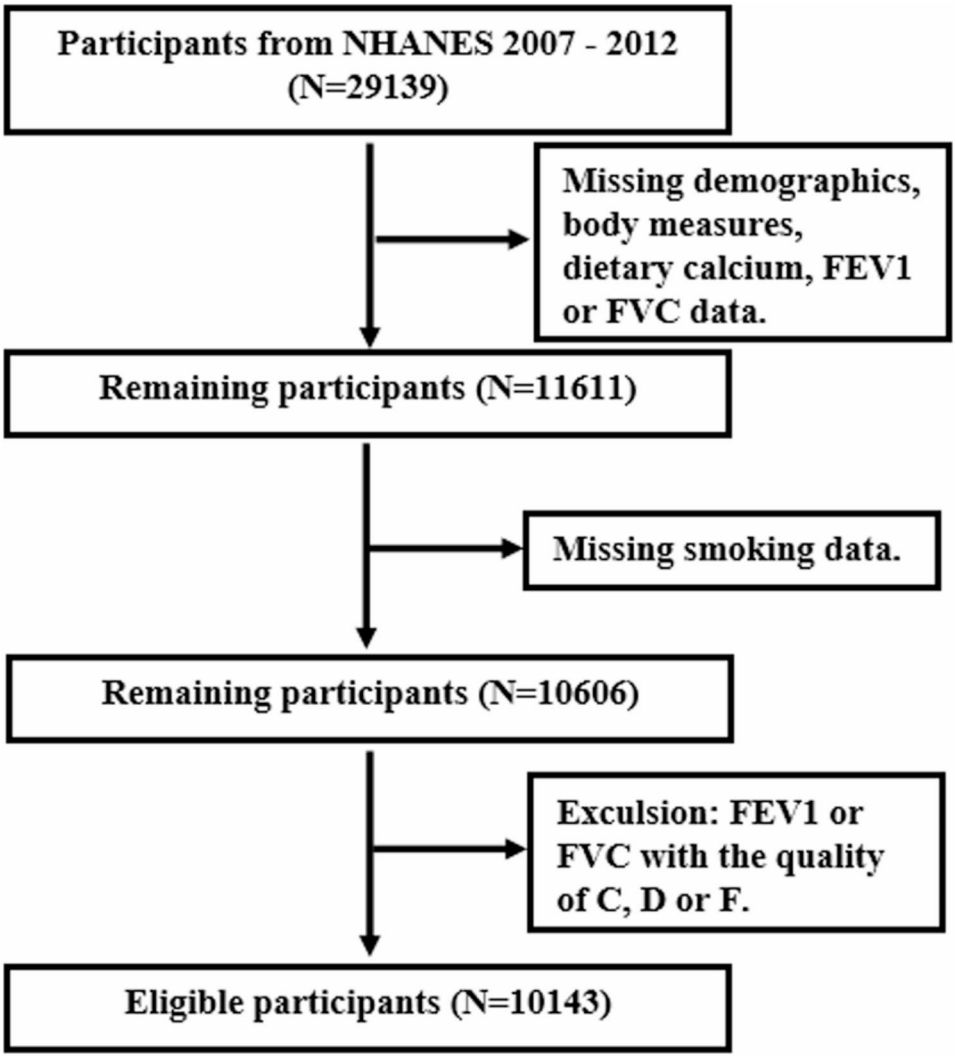

Fig. 1 Flow chart of the screening process. NHANES, National Health and Nutrition Examination Survey; FEV1, forced expiratory volume in 1 s; FVC, forced vital capacity

Statistical analyses

All statistical analyses were analyzed using Statistical Package for the Social Sciences (version 19.0; SPSS Inc., Chicago, IL). Normally distributed data were represented as mean ± standard deviation (SD), and a comparison between groups was performed by *t*-test. Multi-group comparisons were analyzed by one-way ANOVA analysis. Categorical variables were expressed as frequency (percentages) and chi-square tests were used to compare differences between groups. Multivariate logistic regression was performed to assess the effects of dietary calcium intake on CBE and airflow obstruction. Model 1 was adjusted according to age and gender. Model 2 adjusted age, gender, race, and education. Model 3 adjusted age, gender, race, education, ratio of family income to poverty, and marital status. Model 4 adjusted age, gender, race, education, ratio of family income to poverty, marital status, BMI, smoking, dietary energy and dietary protein intake. Linear regression models were applied to explore the relationship between dietary calcium intake and lung function. The *P*-value ˂ 0.05 is considered statistically significant.

Results

Demographic characteristics

A total of 10,143 participants were enrolled in the study, including 594 CBE and 9549 non-CBE individuals (Table 1). The dietary calcium intake was ranged from 29 to 8168 mg/day. The mean age of the CBE group (52.4 ± 15.4 years) was significantly higher than that of the non-CBE group (45.7 ± 16.0 years). Significant differences were found in gender, race, education, ratio of family income to poverty, marital status, and BMI between the two groups. Participants with CBE had a higher proportion of previous smoking (67.0 vs. 44.1%), lower FEV1 (2.5 vs. 3.1 L), lower FVC (3.4 vs. 4.0 L), and

Table 1 Basic characteristics of the study population

Variables	CBE (*N* = 594)	No CBE (*N* = 9549)	*P*-Value
Age (years)	52.4 ± 15.4	45.7 ± 16.0	< 0.001
Gender			< 0.001
Male	220 (37.0%)	4746 (49.7%)	
Female	374 (63.0%)	4803 (50.3%)	
Race			< 0.001
Mexican American	41 (6.9%)	1532 (16.0%)	
Other Hispanic	49 (8.2%)	973 (10.2%)	
Non-Hispanic White	370 (62.3%)	4332 (45.4%)	
Non-Hispanic Black	111 (18.7%)	1939 (20.3%)	
Other Race	23 (3.9%)	773 (8.1%)	
Education			< 0.001
Less than 9th grade	47 (7.9%)	793 (8.3%)	
9-11th grade	119 (20.0%)	1373 (14.4%)	
High school graduate	150 (25.3%)	2116 (22.2%)	
Some college or AA degree	193 (32.5%)	2875 (30.1%)	
College graduate or above	85 (14.3%)	2392 (25.0%)	
Ratio of family income to poverty			< 0.001
≤ 1.00	162 (27.3%)	1875 (19.6%)	
1.01-2.00	192 (32.3%)	2403 (25.2%)	
2.01-4.00	134 (22.6%)	2487 (26.0%)	
> 4.00	106 (17.8%)	2784 (29.2%)	
Marital status			< 0.001
Married	279 (47.0%)	5074 (53.1%)	
Widowed	46 (7.7%)	417 (4.4%)	
Divorced	113 (19.0%)	1046 (11.0%)	
Separated	31 (5.2%)	306 (3.2%)	
Never married	86 (14.5%)	1907 (20.0%)	
Living with partner	39 (6.6%)	799 (8.4%)	
BMI (kg/m^2)			< 0.001
≤ 18.50	15 (2.5%)	111 (1.2%)	
18.51-25	118 (19.9%)	2665 (27.9%)	
25.01-30	167 (28.1%)	3213 (33.6%)	
> 30	294 (49.5%)	3560 (37.3%)	
Smoking			< 0.001
Yes	398 (67.0%)	4215 (44.1%)	
No	196 (33.0%)	5334 (55.9%)	
Dietary calcium intake (mg/day)			0.001
Quartile 1 (≤ 543)	181 (30.5%)	2360 (24.7%)	
Quartile 2 (544–829)	158 (26.6%)	2375 (24.9%)	
Quartile 3 (830–1199)	117 (19.7%)	2419 (25.3%)	
Quartile 4 (> 1200)	138 (23.2%)	2395 (25.1%)	
FEV1 (L)	2.6 ± 0.9	3.1 ± 0.9	< 0.001
FVC (L)	3.4 ± 1.0	4.0 ± 1.1	< 0.001

CBE, chronic bronchitis and emphysema; BMI, body mass index; FEV1, forced expiratory volume in 1 s; FVC, forced vital capacity

a higher proportion of the lowest quartile of dietary calcium intake (30.5 vs. 24.7%). The average intake of dietary calcium was 908.5 ± 636.1 mg/day in the CBE group and 951.9 ± 599.7 mg/day in the non-CBE group.

Our study included 1318 subjects with airflow obstruction and 8825 individuals without airflow obstruction (Table 2). The mean age of the airflow obstruction group

Table 2 Participant characteristics and airflow obstruction

Variables	Airflow obstruction (*N* = 1318)	No airflow obstruction (*N* = 8825)	*P*-Value
Age (years)	58.0 ± 14.3	44.3 ± 15.6	< 0.001
Gender			< 0.001
Male	809 (61.4%)	4157 (47.1%)	
Female	509 (38.6%)	4668 (52.9%)	
Race			< 0.001
Mexican American	90 (6.8%)	1483 (16.8%)	
Other Hispanic	84 (6.4%)	938 (10.6%)	
Non-Hispanic White	841 (63.8%)	3861 (43.8%)	
Non-Hispanic Black	247 (18.7%)	1803 (20.4%)	
Other Race	56 (4.2%)	740 (8.4%)	
Education			< 0.001
Less than 9th grade	124 (9.4%)	716 (8.1%)	
9-11th grade	239 (18.1%)	1253 (14.2%)	
High school graduate	339 (25.7%)	1927 (21.8%)	
Some college or AA degree	339 (25.7%)	2729 (30.9%)	
College graduate or above	277 (21.0%)	2200 (24.9%)	
Ratio of family income to poverty			0.302
≤ 1.00	254 (19.3%)	1783 (20.2%)	
1.01-2.00	340 (25.8%)	2255 (25.6%)	
2.01-4.00	366 (27.8%)	2255 (25.6%)	
> 4.00	358 (27.2%)	2532 (28.7%)	
Marital status			< 0.001
Married	763 (57.9%)	4590 (52.0%)	
Widowed	114 (8.6%)	349 (4.0%)	
Divorced	202 (15.3%)	957 (10.8%)	
Separated	40 (3.0%)	297 (3.4%)	
Never married	106 (8.0%)	1887 (21.4%)	
Living with partner	93 (7.1%)	745 (8.4%)	
BMI (kg/m^2)			< 0.001
≤ 18.50	28 (2.1%)	98 (1.1%)	
18.51-25	446 (33.8%)	2337 (26.5%)	
25.01-30	461 (35.0%)	2919 (33.1%)	
> 30	383 (29.1%)	3471 (39.3%)	
Smoking			< 0.001
Yes	928 (70.4%)	3685 (41.8%)	
No	390 (29.6%)	5140 (58.2%)	
Dietary calcium intake (mg/day)			0.021
Quartile 1 (≤ 543)	375 (28.5%)	2166 (21.4%)	
Quartile 2 (544–829)	323 (24.5%)	2210 (25.0%)	
Quartile 3 (830–1199)	311 (23.6%)	2225 (25.2%)	
Quartile 4 (> 1200)	309 (23.4%)	2224 (25.2%)	
FEV1 (L)	2.5 ± 0.8	3.2 ± 0.9	< 0.001
FVC (L)	3.9 ± 1.2	4.0 ± 1.1	0.013

BMI, body mass index; FEV1, forced expiratory volume in 1 s; FVC, forced vital capacity

was significantly higher than that of the non-airflow obstruction group (58.0 vs. 44.3 years). The ratio of family income to poverty was similar between the two groups ($P = 0.302$). There were significant differences between the airflow obstruction and non-airflow obstruction group in gender, race, education, marital status, and BMI. The proportion of the lowest quartile of dietary calcium intake in the airflow obstruction group was significantly higher than that in the non-airflow obstruction group (28.5 vs. 21.4%). Individuals with airflow obstruction tended to have lower FEV1 (2.5 vs. 3.2 L), lower FVC (3.9 vs. 4.0 L), and a higher proportion of smokers (70.4% vs. 41.8%). The average intake of dietary calcium was lower in the airflow obstruction group (921.2 ± 600.1 mg/day) than that in the non-airflow obstruction group (953.6 ± 602.2 mg/day).

Association between dietary calcium and CBE

When using the lowest quartile of dietary calcium intake as a reference, the second, third, and fourth quartiles of dietary calcium intake reduced the risk of CBE by 0.803 [95% confidence interval (CI): 0.802–0.804; $P < 0.001$], 0.659 (95% CI: 0.659–0.660; $P < 0.001$) and 0.644 (95% CI: 0.643–0.644; $P < 0.001$) times, respectively (Table 3). Dietary calcium intake was negatively related to the risk of CBE ($P < 0.001$) when setting dietary calcium intake as the continuous variable. Increased dietary calcium intake was still correlated with reduced risk of CBE after adjustment of age, gender, race, education, ratio of family income to poverty, marital status, BMI, smoking, dietary energy and dietary protein intake ($P < 0.001$).

Table 3 Multivariate weighted regression analysis of association between dietary calcium intake and CBE

Model	Dietary calcium intake (mg/day)	OR	95% CI	*P*-Value
Unadjusted model	≤ 543	1.0 (Reference)	1.0 (Reference)	
	544–829	0.803	0.802–0.804	< 0.001
	830–1199	0.659	0.659–0.660	< 0.001
	> 1200	0.644	0.643–0.644	< 0.001
Adjusted model 1[a]	≤ 543	1.0 (Reference)	1.0 (Reference)	
	544–829	0.825	0.824–0.826	< 0.001
	830–1199	0.723	0.722–0.724	< 0.001
	> 1200	0.797	0.796–0.798	< 0.001
Adjusted model 2[b]	≤ 543	1.0 (Reference)	1.0 (Reference)	
	544–829	0.887	0.886–0.888	< 0.001
	830–1199	0.794	0.793–0.795	< 0.001
	> 1200	0.891	0.890–0.892	< 0.001
Adjusted model 3[c]	≤ 543	1.0 (Reference)	1.0 (Reference)	
	544–829	0.907	0.906–0.908	< 0.001
	830–1199	0.829	0.828–0.830	< 0.001
	> 1200	0.917	0.916–0.918	< 0.001
Adjusted model 4[d]	≤ 543	1.0 (Reference)	1.0 (Reference)	
	544–829	0.922	0.921–0.923	< 0.001
	830–1199	0.848	0.847–0.849	< 0.001
	> 1200	0.936	0.935–0.937	< 0.001

[a] Adjusted for age and gender

[b] Adjusted for age, gender, race and education

[c] Adjusted for age, gender, race, education, ratio of family income to poverty and marital status

[d] Adjusted for age, gender, race, education, ratio of family income to poverty, marital status, BMI, smoking, dietary energy and dietary protein intake

CBE, chronic bronchitis and emphysema; OR, odds ratio; CI, confidence interval; BMI, body mass index

Stratified analysis was performed based on categories of age, gender and smoking. The association between higher dietary calcium intake and lower risk of CBE still existed in men, women, subgroup of age ≤ 40 or > 40, smokers and non-smokers (Table 4). When compared to the first quartile, the fourth quartile of dietary calcium intake reduced the risk of CBE by 0.460 (95% CI: 0.459 to 0.460; $P < 0.001$) times in non-smoking participants.

Association between dietary calcium intake and lung function

Multivariate weighted regression analysis showed that the second, third, and fourth quartiles reduced the risk of airflow obstruction by 0.923 (95% CI: 0.922–0.924; $P < 0.001$), 0.785 (95% CI: 0.785–0.786; $P < 0.001$) and 0.743 (95% CI: 0.742–0.743; $P < 0.001$) times, respectively (Table 5). Dietary calcium intake was negatively correlated with the risk of airflow obstruction ($P < 0.001$) when setting dietary calcium intake as the continuous variable. Dietary calcium intake remained significant with the risk of airflow obstruction after adjusting for all covariates of interest (model 4) ($P < 0.001$).

The FEV1 in subjects with the first, second, third, and fourth quartiles of dietary calcium intake was 2.8 ± 0.8, 3.0 ± 0.9, 3.1 ± 0.9, and 3.4 ± 0.9 L, respectively ($P < 0.001$, Table 6). This significant difference persisted among all subgroups after stratified analyses of CBE and airflow obstruction. Multiple linear regression suggests that dietary calcium intake positively predicts FEV1 ($\beta = 0.225$, $P < 0.001$) when setting dietary calcium intake as the categorical variable. Elevated dietary calcium intake was still correlated with increased measures of FEV1 after adjustment of age, gender, race, education, ratio of family income to poverty, marital status, BMI, smoking, dietary energy and dietary protein intake ($\beta = 0.052$, $P < 0.001$). We also set dietary calcium intake as a continuous variable, and found that the dietary calcium intake was positively correlated with FEV1 ($\beta = 0.236$, $P < 0.001$; Fig. 2). The significant difference was robust after covariate adjustment ($\beta = 0.053$, $P < 0.001$).

The FVC of individuals in the first, second, third, and fourth quartiles of dietary calcium intake was 3.6 ± 1.0, 3.8 ± 1.0, 4.0 ± 1.1, and 4.3 ± 1.1 L, respectively ($P < 0.001$, Table 6). Dietary calcium intake remained significant with FVC among all subgroups after stratified analyses

Table 4 Subgroup analysis of gender, age and smoking status about associations between dietary calcium intake and CBE

Characteristic	Calcium intake (mg/day)	Unadjusted model			Adjusted model [a]		
		OR	95% CI	*P*-Value	OR	95% CI	*P*-Value
Gender							
Male	≤ 543	1.0 (Reference)	1.0 (Reference)		1.0 (Reference)	1.0 (Reference)	
	544–829	0.890	0.889–0.892	< 0.001	1.024	1.022–1.026	< 0.001
	830–1199	0.952	0.951–0.954	< 0.001	1.203	1.201–1.206	< 0.001
	> 1200	0.604	0.603–0.606	< 0.001	0.854	0.852–0.856	< 0.001
Female	≤ 543	1.0 (Reference)	1.0 (Reference)		1.0 (Reference)	1.0 (Reference)	
	544–829	0.800	0.799–0.801	< 0.001	0.886	0.884	< 0.001
	830–1199	0.555	0.555–0.556	< 0.001	0.674	0.673	< 0.001
	> 1200	0.847	0.846–0.848	< 0.001	1.052	1.050	< 0.001
Age (years)							
≤ 40	≤ 543	1.0 (Reference)	1.0 (Reference)		1.0 (Reference)	1.0 (Reference)	
	544–829	0.780	0.778–0.782	< 0.001	0.965	0.962–0.967	< 0.001
	830–1199	0.546	0.545–0.547	< 0.001	0.764	0.762–0.766	< 0.001
	> 1200	0.588	0.586–0.589	< 0.001	0.817	0.815–0.820	< 0.001
> 40	≤ 543	1.0 (Reference)	1.0 (Reference)		1.0 (Reference)	1.0 (Reference)	
	544–829	0.820	0.819–0.821	< 0.001	0.930	0.929–0.931	< 0.001
	830–1199	0.713	0.712–0.714	< 0.001	0.885	0.884–0.886	< 0.001
	> 1200	0.733	0.732–0.734	< 0.001	1.018	1.016–1.019	< 0.001
Smoking status							
Yes	≤ 543	1.0 (Reference)	1.0 (Reference)		1.0 (Reference)	1.0 (Reference)	
	544–829	0.869	0.868–0.870	< 0.001	0.986	0.984–0.987	< 0.001
	830–1199	0.708	0.707–0.709	< 0.001	0.870	0.869–0.872	< 0.001
	> 1200	0.849	0.847–0.850	< 0.001	1.120	1.118–1.122	< 0.001
No	≤ 543	1.0 (Reference)	1.0 (Reference)		1.0 (Reference)	1.0 (Reference)	
	544–829	0.750	0.749–0.751	< 0.001	0.845	0.843–0.846	< 0.001
	830–1199	0.635	0.634–0.637	< 0.001	0.826	0.825–0.828	< 0.001
	> 1200	0.460	0.459–0.460	< 0.001	0.707	0.706–0.709	< 0.001

CBE, chronic bronchitis and emphysema; OR, odds ratio; CI, confidence interval

[a] Adjusted for age, gender, race, education, ratio of family income to poverty, marital status, BMI, smoking, dietary energy and dietary protein intake

of CBE and airflow obstruction. Elevated dietary calcium intake was correlated with increased measures of FVC ($\beta = 0.232$, $P < 0.001$) using dietary calcium intake as the categorical variable. Dietary calcium intake positively predicts FVC after adjustment of all confounders of interest ($\beta = 0.050$, $P < 0.001$). We also set the dietary calcium intake as a continuous variable, and found that dietary calcium intake was positively related to FVC ($\beta = 0.252$, $P < 0.001$; Fig. 2). The significant difference was robust after covariate adjustment ($\beta = 0.060$, $P < 0.001$).

Discussion

COPD is a respiratory disorder characterized by chronic airflow obstruction secondary to chronic bronchitis, emphysema, or both [19]. Calcium is an essential mineral element for human nutrition. However, the relationship between dietary calcium intake and CBE was not fully understood. Our present study showed that increased dietary calcium intake was correlated with reduced risk of CBE in American population. Dietary calcium intake reduced the risk of airflow obstruction. A higher intake of dietary calcium was associated with significantly higher measures of FEV1 and FVC.

Calcium intake is often related to the intake of foods such as milk, cheese, and yogurt, as dairy products are rich sources of calcium [20]. Although studies on the effects of calcium have focused primarily on bone health, the role of dietary calcium has recently shifted to other health conditions [21]. Cellular calcium ion is associated with the contraction of airway smooth muscle cells. The regulation of calcium ion in airway smooth muscle is related to COPD [18]. In the present study, the average intake of dietary calcium was 908.5 ± 636.1 mg/day in the CBE group and 951.9 ± 599.7 mg/day in the non-CBE group. Increased dietary calcium intake was still correlated with reduced risk of CBE in American participants after adjustment of age, gender, race, education, ratio of family income to poverty, marital status, BMI, smoking, dietary energy and dietary protein intake. These results are consistent with the findings of Hirayama and colleagues [22] who found an inverse association between dietary calcium intake and COPD risk in Japanese adults. However, in their study, the average intake of

Table 5 Multivariate weighted regression analysis of association between dietary calcium intake and airflow obstruction

Model	Dietary calcium intake (mg/day)	OR	95% CI	*P*-Value
Unadjusted model	≤ 543	1.0 (Reference)	1.0 (Reference)	
	544–829	0.923	0.922–0.924	< 0.001
	830–1199	0.785	0.785–0.786	< 0.001
	> 1200	0.743	0.742–0.743	< 0.001
Adjusted model 1[a]	≤ 543	1.0 (Reference)	1.0 (Reference)	
	544–829	0.836	0.835–0.836	< 0.001
	830–1199	0.827	0.827–0.828	< 0.001
	> 1200	0.898	0.897–0.899	< 0.001
Adjusted model 2[b]	≤ 543	1.0 (Reference)	1.0 (Reference)	
	544–829	0.928	0.927–0.929	< 0.001
	830–1199	0.862	0.862–0.863	< 0.001
	> 1200	0.881	0.880–0.882	< 0.001
Adjusted model 3[c]	≤ 543	1.0 (Reference)	1.0 (Reference)	
	544–829	0.929	0.928–0.930	< 0.001
	830–1199	0.863	0.863–0.864	< 0.001
	> 1200	0.882	0.881–0.882	< 0.001
Adjusted model 4[d]	≤ 543	1.0 (Reference)	1.0 (Reference)	
	544–829	0.921	0.921–0.922	< 0.001
	830–1199	0.791	0.790–0.792	< 0.001
	> 1200	0.812	0.812–0.813	< 0.001

[a] Adjusted for age and gender

[b] Adjusted for age, gender, race and education

[c] Adjusted for age, gender, race, education, ratio of family income to poverty and marital status

[d] Adjusted for age, gender, race, education, ratio of family income to poverty, marital status, BMI, smoking, dietary energy and dietary protein intake

OR, odds ratio; CI, confidence interval

dietary calcium in the COPD and non-COPD group was 463 ± 210 and 545 ± 245 mg/day, respectively.

COPD is a preventible condition characterized by progressive airflow obstruction [23]. According to the GOLD 2025 criteria, the presence of a pre-bronchodilator FEV1/FVC < 0.7 was used to assess whether there is airflow obstruction in symptomatic patients. If the prebronchodilator spirometry does not show airflow obstruction, post-bronchodilator spirometry is not required unless there is a very high clinical suspicion of COPD [24]. In the present study, the average intake of dietary calcium was lower in the airflow obstruction group (921.2 ± 600.1 mg/day) than that in the non-airflow obstruction group (953.6 ± 602.2 mg/day). Dietary calcium intake remained significant with the risk of airflow obstruction after adjusting for all covariates of interest. The mechanism by which dietary calcium intake affects airflow obstruction is still unclear. Small airway diseases in patients with COPD cause luminal stenosis, which further lead to increased airway resistance and airflow obstruction. Small airway diseases include airway remodelling, mucus overproduction and airway inflammation [7–8]. Airway epithelial cells release a series of mediators that regulate immune responses and airway inflammation when exposed to harmful environmental stimuli. A key cellular mechanism in airway inflammation is calcium signaling, which stimulates the airway epithelium to produce and release prostaglandins, cytokines and chemokines [25]. Further research is needed to explore in detail the role of calcium ions in airway smooth muscle contraction, inflammatory responses, and determine how calcium intake may affect lung function and airflow obstruction by influencing these physiologic processes.

The assessment of FEV1 is critical in estimating the severity of airflow obstruction and guiding the clinical management of COPD [26]. FEV1 also provides a way to classify different degrees of COPD severity [27]. In the present study, the FEV1 in subjects with the first, second, third, and fourth quartiles of dietary calcium intake was 2.8 ± 0.8, 3.0 ± 0.9, 3.1 ± 0.9, and 3.4 ± 0.9 L, respectively.

Table 6 Associations between dietary calcium intake and lung function

Variables		Dietary calcium intake (mg/day)				*P*-Value
		≤ 543	544–829	830–1199	> 1200	
FEV1(L)	Entire population	2.8 ± 0.8	3.0 ± 0.9	3.1 ± 0.9	3.4 ± 0.9	< 0.001
	CBE					
	Yes	2.4 ± 0.8	2.4 ± 0.8	2.6 ± 0.9	2.7 ± 0.9	0.001
	No	2.8 ± 0.8	3.0 ± 0.8	3.2 ± 0.9	3.4 ± 0.9	< 0.001
	Airflow obstruction					
	Yes	2.2 ± 0.7	2.4 ± 0.8	2.5 ± 0.8	2.8 ± 0.9	< 0.001
	No	2.9 ± 0.8	3.1 ± 0.8	3.2 ± 0.9	3.5 ± 0.9	< 0.001
FVC (L)	Entire population	3.6 ± 1.0	3.8 ± 1.0	4.0 ± 1.1	4.3 ± 1.1	< 0.001
	CBE					
	Yes	3.3 ± 1.0	3.3 ± 1.0	3.6 ± 1.1	3.7 ± 1.1	0.001
	No	3.6 ± 1.0	3.9 ± 1.0	4.0 ± 1.0	4.4 ± 1.1	< 0.001
	Airflow obstruction					
	Yes	3.5 ± 1.0	3.8 ± 1.1	3.9 ± 1.2	4.4 ± 1.2	< 0.001
	No	3.6 ± 1.0	3.8 ± 1.0	4.0 ± 1.1	4.3 ± 1.1	< 0.001

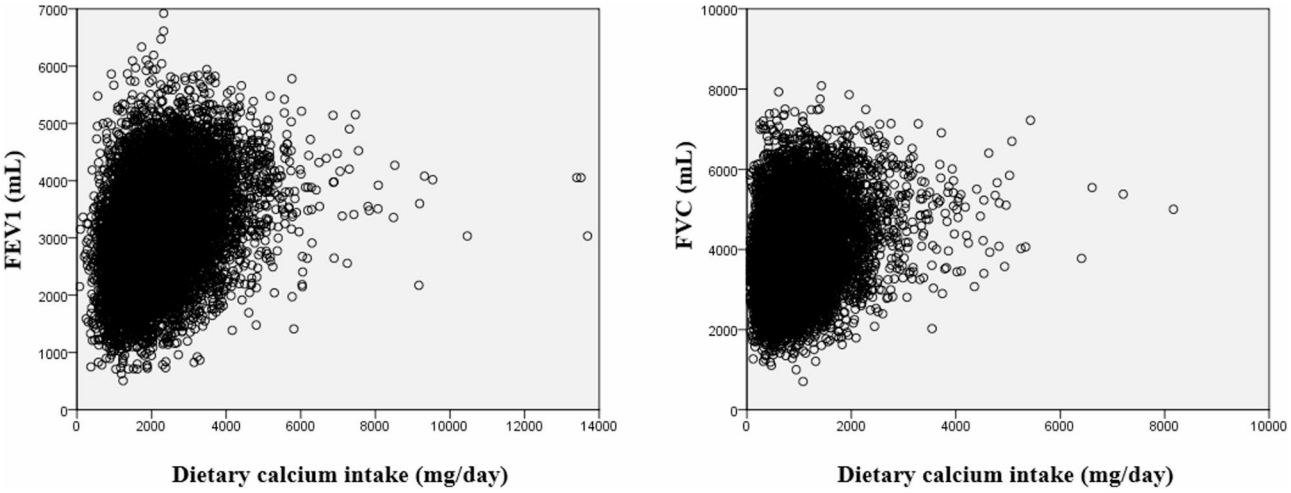

Fig. 2 Associations of dietary calcium intake with FEV1 and FVC. FEV1, forced expiratory volume in 1 s; FVC, forced vital capacity

Multiple linear regression suggests that dietary calcium intake positively predicts FEV1. Our findings are similar to those of Hirayama et al. [22] who found that the FEV1 was positively correlated with dietary calcium intake.

The present study has several limitations. Firstly, since the cross-sectional design was performed in the NHANES database, we were not able to determine the causality. Moreover, the observed associations may be influenced by confounding factors, such as overall diet quality, physical activity, osteoporosis treatment or underlying health conditions, which were not fully accounted for in current analysis [28]. Secondly, the evaluation of 24-h dietary intake may not truly reflect actual intake. Dietary supplements were not taken into account. Additionally, dietary calcium intake was self-reported, which introduces the possibility of recall bias and measurement error [29]. However, 24-h dietary recall interview is widely regarded as the most appropriate way to estimate the average intake at the population level [30–31]. Thirdly, the definition of CBE was depended on questionnaire-based surveys and self-reports by participants. It might result in some bias in present study. However, similar method was employed in the previous studies using NHANES data [32–35]. Fourthly, the data of our study were obtained from the NHANES database applicable to American participants. The generalizability of the findings may be limited to populations with similar dietary patterns and lifestyles as those in the NHANES dataset. Thus, our findings need to be validated in other populations. Fifthly, the dietary calcium intake was ranged from 29 to 8168 mg/day in the present study. Further studies are needed to investigate the effects of dietary calcium on CBE if the intake exceeds this range.

Conclusions

Our study suggests that the dietary calcium intake is negatively correlated with the risk of CBE in American population. Participants with CBE have higher dietary calcium intakes as compared to individuals without CBE. Higher intakes of dietary calcium were associated with lower risk of airflow obstruction, and higher measures of FEV1 and FVC. However, the biological mechanism of calcium intake affecting lung function or CBE risk remains to be further studied. Moreover, the cross-sectional design, potential confounding factors, and lack of mechanistic insight highlight the need for further research, particularly longitudinal and interventional studies, to confirm our findings, determine optimal calcium intake for reducing CBE risk and explore the underlying mechanisms. If validated, these results could have important implications for dietary recommendations aimed at improving respiratory health in individuals at risk of CBE.

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Author contributions

XF.L and W.Y. wrote the main manuscript text. XF.L and W.Y. prepared figure 1; Tables 1, 2, 3, 4, 5 and 6. All authors reviewed the manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Ethics approval was accepted by National Center for Health Statistics Research Ethics Review Board, and all participants provided informed consent.

Consent to publish
Not applicable.

Disclosure
Not applicable.

Competing interests
The authors declare no competing interests.

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References

1. Wang Y, Yu Y, Zhang X, Zhang H, Zhang Y, Wang S, Yin L. Combined association of urinary volatile organic compounds with chronic bronchitis and emphysema among adults in NHANES 2011–2014: the mediating role of inflammation. Chemosphere. 2024;361:141485.
2. Han MK, Hogg J, Humphries S, Lee KS, Lynch D, Machnicki S, Mehta A, Mina B, Naidich D, Naidich J, Naqvi Z, Ohno Y, Regan E, Travis WD, Washko G, Braman S. Lung imaging in COPD part 1: clinical usefulness. Chest. 2023;164(1):69–84.
3. Holtjer JCS, Bloemsma LD, Beijers RJHCG, Cornelissen MEB, Hilvering B, Houweling L, Vermeulen RCH, Downward GS. Maitland-Van der Zee AH; P4O2 consortium. Identifying risk factors for COPD and adult-onset asthma: an umbrella review. Eur Respir Rev. 2023;32(168):230009.
4. Islam F, Muni M, Mitra S, Emran TB, Chandran D, Das R, Rauf A, Safi SZ, Chidambaram K, Dhawan M, Cheon C, Kim B. Recent advances in respiratory diseases: dietary carotenoids as choice of therapeutics. Biomed Pharmacother. 2022;155:113786.
5. Zhai H, Wang Y, Jiang W. Fruit and vegetable intake and the risk of chronic obstructive pulmonary disease: A Dose-Response Meta-Analysis of observational studies. Biomed Res Int. 2020;2020:3783481.
6. Won HK, Song WJ, Moon SD, Sohn KH, Kim JY, Kim BK, Park HW, Bachert C, Cho SH. Staphylococcal Enterotoxin-Specific IgE sensitization: A potential predictor of fixed airflow obstruction in elderly asthma. Allergy Asthma Immunol Res. 2023;15(2):160–73.
7. Liu C, Li P, Zheng J, Wang Y, Wu W, Liu X. Role of necroptosis in airflow limitation in chronic obstructive pulmonary disease: focus on small-airway disease and emphysema. Cell Death Discov. 2022;8(1):363.
8. Santus P, Radovanovic D, Pecchiari M, Ferrando M, Tursi F, Patella V, Braido F. The relevance of targeting treatment to small airways in asthma and COPD. Respir Care. 2020;65(9):1392–412.
9. Huang WJ, Fan XX, Yang YH, Zeng YM, Ko CY. A review on the role of oral nutritional supplements in chronic obstructive pulmonary disease. J Nutr Health Aging. 2022;26(7):723–31.
10. Rondanelli M, Faliva MA, Peroni G, Infantino V, Gasparri C, Iannello G, Perna S, Alalwan TA, Al-Thawadi S, Corsico AG. Food pyramid for subjects with chronic obstructive pulmonary diseases. Int J Chron Obstruct Pulmon Dis. 2020;15:1435–48.
11. Hua Y, Liu HL, Sun JY, Kong XQ, Sun W, Xiong YQ. Association between serum calcium and the prevalence of hypertension among US adults. Front Cardiovasc Med. 2021;8:719165.
12. Mekal D, Czerw A, Deptala A. Dietary behaviour and nutrition in patients with COPD treated with Long-Term oxygen therapy. Int J Environ Res Public Health. 2021;18(23):12793.
13. Cruz-Pierard SM, Nestares T, Amaro-Gahete FJ. Vitamin D and calcium as key potential factors related to colorectal cancer prevention and treatment: A systematic review. Nutrients. 2022;14(22):4934.
14. Bargagli M, Ferraro PM, Vittori M, Lombardi G, Gambaro G, Somani B. Calcium and vitamin D supplementation and their association with kidney stone disease: A narrative review. Nutrients. 2021;13(12):4363.
15. Vlok M, Snoddy AME, Ramesh N, Wheeler BJ, Standen VG, Arriaza BT. The role of dietary calcium in the etiology of childhood rickets in the past and the present. Am J Hum Biol. 2023;35(2):e23819.
16. Rana ZH, Bourassa MW, Gomes F, Khadilkar A, Mandlik R, Owino V, Pettifor JM, Roth DE, Shlisky J, Thankachan P, Weaver CM. Calcium status assessment at the population level: candidate approaches and challenges. Ann N Y Acad Sci. 2022;1517(1):93–106.
17. Rimessi A, Vitto VAM, Patergnani S, Pinton P. Update on calcium signaling in cystic fibrosis lung disease. Front Pharmacol. 2021;12:581645.
18. Du X, Zhi J, Yang D, Wang Q, Luo X, Deng X. Research progress in the mechanism of calcium ion on contraction and relaxation of airway smooth muscle cells. J Recept Signal Transduct Res. 2021;41(2):117–22.
19. Raoof S, Shah M, Make B, Allaqaband H, Bowler R, Fernando S, Greenberg H, Han MK, Hogg J, Humphries S, Lee KS, Lynch D, Machnicki S, Mehta A, Mina B, Naidich D, Naidich J, Naqvi Z, Ohno Y, Regan E, Travis WD, Washko G, Braman S. Lung imaging in COPD part 1: clinical usefulness. Chest. 2023;164(1):69–84.
20. Cormick G, Belizán JM. Calcium Intake Health Nutrients. 2019;11(7):1606.
21. Chen YY, Chen YJ. The relationship between dietary calcium and Age-Related macular degeneration. Nutrients. 2023;15(3):671.
22. Hirayama F, Lee AH, Oura A, Mori M, Hiramatsu N, Taniguchi H. Dietary intake of six minerals in relation to the risk of chronic obstructive pulmonary disease. Asia Pac J Clin Nutr. 2010;19(4):572–7.
23. Seyedrezazadeh E, Moghaddam MP, Ansarin K, Asghari Jafarabadi M, Sharifi A, Sharma S, Kolahdooz F. Dietary factors and risk of chronic obstructive pulmonary disease: a systemic review and Meta-Analysis. Tanaffos. 2019;18(4):294–309.
24. Venkatesan P. GOLD COPD report: 2025 update. Lancet Respir Med. 2024 Dec 5:S2213-2600(24)00413-2. Epub ahead of print.
25. Jairaman A, Prakriya M. Calcium signaling in airway epithelial cells: current Understanding and implications for inflammatory airway disease. Arterioscler Thromb Vasc Biol. 2024;44(4):772–83.
26. Kakavas S, Kotsiou OS, Perlikos F, Mermiri M, Mavrovounis G, Gourgoulianis K, Pantazopoulos I. Pulmonary function testing in COPD: looking beyond the curtain of FEV1. NPJ Prim Care Respir Med. 2021;31(1):23.
27. Agustí A, Celli BR, Criner GJ, Halpin D, Anzueto A, Barnes P, Bourbeau J, Han MK, Martinez FJ, Montes de Oca M, Mortimer K, Papi A, Pavord I, Roche N, Salvi S, Sin DD, Singh D, Stockley R, López Varela MV, Wedzicha JA, Vogelmeier CF. Global initiative for chronic obstructive lung disease 2023 report: GOLD executive summary. Eur Respir J. 2023;61(4):2300239.
28. Huang AA, Huang SY. Shapely additive values can effectively visualize pertinent covariates in machine learning when predicting hypertension. J Clin Hypertens (Greenwich). 2023;25(12):1135–44.
29. Huang AA, Huang SY. Use of machine learning to identify risk factors for insomnia. PLoS ONE. 2023;18(4):e0282622.
30. Zheng X, Hur J, Nguyen LH, Liu J, Song M, Wu K, Smith-Warner SA, Ogino S, Willett WC, Chan AT, Giovannucci E, Cao Y. Comprehensive assessment of diet quality and risk of precursors of Early-Onset colorectal cancer. J Natl Cancer Inst. 2021;113(5):543–52.
31. Wen J, Gu S, Wang X, Qi X. Associations of adherence to the DASH diet and the mediterranean diet with chronic obstructive pulmonary disease among US adults. Front Nutr. 2023;10:1031071.
32. Fei Q, Weng X, Liu K, Liu S, Chen J, Guo X, Jing C. The relationship between metal exposure and chronic obstructive pulmonary disease in the general US population: NHANES 2015–2016. Int J Environ Res Public Health. 2022;19(4):2085.
33. Liu H, Tan X, Liu Z, Ma X, Zheng Y, Zhu B, Zheng G, Hu Y, Fang L, Hong G. Association between Diet-Related inflammation and COPD: findings from NHANES III. Front Nutr. 2021;8:732099.
34. Yentes JM, Sayles H, Meza J, Mannino DM, Rennard SI, Stergiou N. Walking abnormalities are associated with COPD: an investigation of the NHANES III dataset. Respir Med. 2011;105(1):80–7.
35. Wang Y, Meng Z, Wei S, Li X, Su Z, Jiang Y, Wu H, Pan H, Wang J, Zhou Q, Qiao Y, Fan Y. Urinary volatile organic compound metabolites and COPD among US adults: mixture, interaction and mediation analysis. Environ Health. 2024;23(1):45.

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