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Published In
Int. J. Epidemiol, 33, 271-278.

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IJE vol.33 no.2 © International Epidemiological Association 2004; all rights reserved.
*International Journal of Epidemiology* 2004;**33**:271–278
DOI: 10.1093/ije/dyh003

# A matter of life and breath: childhood socioeconomic status is related to young adult pulmonary function in the CARDIA study

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**Accepted** 16 July 2003

**Background** Socioeconomic status (SES) may contribute to the trajectory of pulmonary function over the life course. Studies suggest that people with lower (versus higher) SES during childhood subsequently have lower levels of adult pulmonary function. But prospective studies are sparse across young adulthood, an important phase in pulmonary development.

**Methods** Participants were from the Coronary Artery (Disease) Risk Development in (Young) Adults (CARDIA) study: 5113 young adults ages 18–30 at baseline, approximately balanced within centres across gender, self-identified race/ethnicity (Black, White), and current SES. Childhood SES was ascertained from baseline self-reports of parents' highest completed education. Pulmonary function in young adulthood was measured using $FEV_1$ (forced expiratory volume in one second) and FVC (forced vital capacity), assessed on three occasions over a period of 5 years.

**Results** Longitudinal analyses suggested that rates of change in both $FEV_1$ and FVC differed in a gradient fashion by childhood SES. As shown by significant childhood SES by time interaction terms, these associations with $FEV_1$ were robust for men ($b = 1.59E{-}3$, $SE = 5.21E{-}4$, $P < 0.001$) and women ($b = 1.93E{-}3$, $SE = 4.80E{-}4$, $P < 0.001$), and adjusted for multiple potential confounders including smoking. Results were similar for FVC. Subsequent examination of the interaction terms suggested that $FEV_1$ and FVC declined for participants in the lowest childhood SES group, showed continued plateau or growth for those in the highest group, and were intermediate for the middle group.

**Conclusions** Childhood SES may influence men's and women's young adult pulmonary function in two ways. First, individuals with lower childhood SES may not attain as high levels of pulmonary function in early adulthood relative to individuals with higher childhood SES. Second, pulmonary function may decline earlier and faster for individuals with lower childhood SES.

**Keywords** Pulmonary function tests, forced expiratory volume, vital capacity, socioeconomic status, adults, social medicine

† We ran analyses both with childhood SES as a continuous and as a class variable, and found essentially the same results. To conserve parameters we used linear childhood SES and due to space constraints reported only the models with linear childhood SES variables.

‡ For interpretability of the projections, we adjusted for height$^2$ as a covariate, instead of dividing pulmonary function by height$^2$ and taking the log-transformation. All other parameters used in the models for the projections are as in Model 2 (Tables 2 and 3).

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Reduced maximally attained pulmonary function and accelerated rate of pulmonary function decline are risk factors for the development of undesirable health conditions including chronic obstructive pulmonary disease (COPD), cardiovascular disease, and early mortality.[1] COPD, for instance, results in poor physical, psychological, and social functioning,[2] poses a considerable economic burden,[3] and is projected to be the third leading cause of death by 2020.[4] Factors contributing to poor pulmonary function have not yet been elucidated fully.

Researchers have conceptualized socioeconomic factors as a 'fundamental cause' of disease.[5] However, few studies go beyond considering socioeconomic status (SES) as a confounder when examining pulmonary function.[6,7] Those that do suggest low SES is associated with low mean levels of pulmonary function (for a review see ref. 8). Moreover, SES explains pulmonary function differences observed across other social status markers, and beyond effects of smoking.[9] Important gaps remain in research linking SES and pulmonary function.

No studies examine how SES influences the trajectory of pulmonary function in young adulthood. Pulmonary capacity is thought to be dynamic, with growth in childhood and adolescence, a plateau during young adulthood, and decline beginning in later adulthood. A recent study examined how early life factors like asthma contribute to level of pulmonary function and change in a sample of young adults, but this study did not consider SES.[10] Because childhood SES may be linked with differential exposure to a variety of risk factors (e.g. asthma, allergens, tobacco smoke)[11] resulting in childhood respiratory trouble,[12] childhood SES may be a critical predictor of adult pulmonary function.[13] Young adults are important to study because maximally attained pulmonary function is achieved in young adulthood,[14] and higher peak pulmonary capacity serves as a buffer against subsequent decreases in pulmonary function related to ageing and lifetime exposure to environmental toxins. Further, early pulmonary function decline is linked to early mortality. [Wang X *et al.*, Early predictors of chronic obstructive pulmonary disease, submitted manuscript, 2002.]

We examined the association between childhood SES and young adult pulmonary function in the Coronary Artery (Disease) Risk Development in (Young) Adults (CARDIA) study. Based on previous research on SES and health,[8,15,16] we hypothesized a gradient relation between childhood SES and young adult pulmonary function at baseline, and also expected to see this gradient persist over time. Finally, we hypothesized that lower childhood SES would be associated with a faster rate of pulmonary function decline.

# Method

The CARDIA study was designed to assess cardiovascular risk factors in young adults. Public-use data from CARDIA were used for these analyses. The Human Studies Committee of the Brigham and Women's Hospital approved the study. Details about study design and recruitment are available elsewhere.[17]

The study was conducted in four urban centres in the US: Minneapolis, MN; Birmingham, AL; Chicago, IL; and Oakland, CA. The total sample consisted of 5115 participants (2787 women and 2328 men) approximately balanced within each centre across gender, race/ethnicity, and SES. The following participants were included: those who self-identified as Black or as White (US Census Bureau category), with a permanent address in the target area, free of long-term disease or disability, and not pregnant at baseline. Pulmonary function was obtained at baseline (1985–1986), year 2, and year 5. Data on sociodemographic factors, anthropometry, asthma history, and smoking status were also available. In the public-use data on CARDIA, measurements were deleted when they would have enabled identification of participants (e.g. name, birth date) or were deemed too sensitive to distribute (e.g. illicit drug use). Of eligible participants, 50%, ages 18–30 years, participated. Two participants had incomplete data, resulting in 5113 participants for these analyses.

## Measures

### *Childhood SES*

Childhood SES was determined by the highest level of education attained between participants' parents. The range available for father's highest education was 11 (11th grade) to 13 (some college or more). More finely grained distinctions for higher levels of father's education were not available. The range for mother's highest education was 11 (11th grade) to 17 (some graduate school or more). A variable was created taking the highest education completed between the participant's father and mother, with values ranging from 11 (11th grade) to 13 (some college or more).† Historically, parents' level of one or more markers of SES (income, education, occupation, wealth) has been used as a proxy for childhood SES.[18] We used education because it is easily measured, comparable across studies in the US and Western Europe, and a more reliable measure for women than is occupation.[19] If the participant had only one parent, that parent's highest education was used to compute childhood SES. Participants missing childhood SES data for both parents ($n = 318$) were not included.

### *Pulmonary function*

Pulmonary function was assessed with a Collins Survey 8-litre water-sealed spirometer and the Eagle II Microprocessor (Warren E Collins, Inc., Braintree, MA USA) while participants were standing and wearing nose clips. Pulmonary function data were acceptable if at least three reproducible tests of forced expiratory volume in one second ($FEV_1$) and forced vital capacity (FVC) were taken, with up to five attempts, in accordance with American Thoracic Society standards for pulmonary function. Of the 5113 CARDIA participants, 4861 (95%) yielded acceptable data for $FEV_1$ and FVC.

### *Other determinants of pulmonary function*

Baseline height was measured to the nearest 0.5 cm. Age, gender, current SES, history of asthma (unconfirmed and doctor confirmed), parental (maternal and paternal) smoking status, and participant smoking status were ascertained at baseline by an interviewer-administered questionnaire. Current SES was assessed by number of years of education the participant had completed. Unconfirmed asthma symptoms was ascertained by answering yes to wheezing 'occasionally apart from colds' or 'most days or nights,' and to endorsing breathlessness 'when hurrying on the level of walking up a slight hill.' Doctor-confirmed asthma was defined as answering yes to both 'have you ever had asthma?' and 'was it confirmed by a doctor?'

### Analyses

We estimated parameters for the effect of childhood SES on pulmonary function using hierarchical linear modelling (HLM; also known as random effects modelling) using repeated measures analysis in the Statistical Analysis System.[20] This allows examination of how people change over time,[21] and has been used to examine pulmonary change.[22] Multiple observations at different times are viewed as nested within the individual. Each model has two levels: (1) a 'within' subject level that specifies individual time paths, and (2) a 'between' subjects level that considers whether group membership (e.g. low SES versus high SES) accounts for differences in rates of change.[21] After examining variance in individual-level intercepts and slopes, a conditional model predicts intercept and slope terms using group as a predictor variable. These models can accommodate missing values of the dependent variable, and allow control for potential confounding variables and baseline pulmonary function when examining rates of decline. Covariates are set as fixed effects in these analyses. The covariance structure was specified using a compound symmetry model which was the best fitting model using Aikaike's Information Criterion and maximum likelihood ratio tests. As we were primarily interested in the 'between group' effects (i.e. childhood SES levels), we present only the data for fixed effects. Values for $FEV_1$ and FVC were divided by height-squared and log-transformed, which has been shown, in this sample, to be the most effective yet parsimonious adjustment for height.[23] An age-squared term was added to the models, in addition to linear age, to account for non-linear effects (i.e. growth, plateau, and decline).[10] All predictor variables were centred. Thus, the intercept may be interpreted to describe the mean or reference value for each of the other predictor variables. To determine whether childhood SES influenced rate of pulmonary function decline in the mixed regression models, we created an interaction term for childhood SES and time, using linear terms for both.

## Results

Table 1 shows pulmonary function and its determinants by childhood SES. Lower childhood SES individuals had lower current SES, were shorter, older (women only), more likely to report unconfirmed asthma symptoms but less likely to report doctor-confirmed asthma, children of fathers who ever smoked, and current smokers themselves.

Hierarchical linear models were used to examine adjusted baseline levels and rates of decline in pulmonary function over time according to childhood SES. Model 1 used childhood SES to predict pulmonary function, adjusting for standard control variables: baseline pulmonary function, age, and $age^2$. Model 2 included the variables adjusted for in Model 1, and the following covariates: current SES, asthma history (unconfirmed symptoms and confirmed by a doctor), parental smoking (maternal and paternal), and participant smoking status (current and former). Tables 2 and 3 present HLM results for $FEV_1$ and FVC, respectively.

Model 1 suggested a monotonic association between childhood SES and pulmonary function. Participants with higher levels of childhood SES had higher levels of $FEV_1$ (Table 2), as evidenced by a statistically significant main effect of childhood SES for both men ($b = 0.043$, SE = 4.67E−3, $P < 0.001$) and women ($b = 0.033$, SE = 3.85E−3, $P < 0.001$). Similarly,

**Table 1** Descriptive statistics by level of childhood socioeconomic status (SES), for men and women

| | Men | | | Women | | |
|---|---|---|---|---|---|---|
| | 11th grade ($n$ = 208) | 12th grade ($n$ = 864) | At least some college ($n$ = 1108) | 11th grade ($n$ = 330) | 12th grade ($n$ = 942) | At least some college ($n$ = 1343) |
| **Mean pulmonary function** | | | | | | |
| $FEV_1$ (SD) (l) | 4.00 (0.63)[a] | 3.96 (0.66)[a] | 4.27 (0.66)[b] | 2.95 (0.47)[a] | 3.00 (0.50)[a] | 3.15 (0.48)[b] |
| FVC (SD) (l) | 4.84 (0.83)[a] | 4.87 (0.81)[a] | 5.29 (0.83)[b] | 3.48 (0.56)[a] | 3.56 (0.59)[a] | 3.76 (0.60)[b] |
| **Determinants of pulmonary function** | | | | | | |
| Height (SD) (cm) | 176.86 (6.71)[a] | 177.09 (6.67)[a] | 178.25 (6.48)[b] | 163.23 (5.84)[a] | 163.85 (6.46)[a] | 165.05 (6.39)[b] |
| Age (SD) (years) | 25.06 (3.71)[a] | 24.61 (3.69)[a] | 24.71 (3.50)[a] | 25.50 (3.41)[a] | 24.55 (3.76)[b] | 24.71 (3.64)[b] |
| Current SES, in years of education | 12.78 (1.63)[a] | 13.05 (1.75)[b] | 14.24 (1.80)[c] | 12.77 (1.63)[a] | 13.12 (1.69)[b] | 14.38 (1.66)[c] |
| Asthma, unconfirmed (%) [1] | 6.25* | 4.86 | 3.52 | 8.79* | 7.75 | 4.99 |
| Asthma, confirmed by a doctor (%) | 5.77* | 9.06 | 9.87 | 4.86* | 7.26 | 9.35 |
| Maternal smoking (%) | 46.08 [ns] | 45.64 | 48.55 | 50.61 [ns] | 50.74 | 51.30 |
| Paternal smoking (%) | 59.22* | 57.91 | 57.72 | 60.67* | 60.58 | 58.08 |
| Former smokers (%) | 7.32* | 11.28 | 14.99 | 10.91* | 12.91 | 16.07 |
| Current smokers (%) | 43.41* | 35.00 | 24.22 | 36.67* | 32.02 | 23.84 |

$FEV_1$ = forced expiratory volume in one second; FVC = forced vital capacity.

Note. Superscripts of different letters denote statistically significant differences at $P < 0.05$. *N*s across each childhood SES category total 4795 (and not the grand total of 5113) because 318 cases were missing data required to calculate highest combined parent education.

[1] Unconfirmed asthma was ascertained by answering yes to wheezing 'occasionally apart from colds' or 'most days or nights,' and to endorsing breathlessness 'when hurrying on the level of walking up a slight hill.'

*$\chi^2$ test for difference among childhood SES levels for within-gender determinant categories, d.f. = 2, $P < 0.05$.

ns = $\chi^2$ test for difference among childhood SES levels for within-gender determinant categories, d.f. = 2, ns.

**Table 2** Childhood socioeconomic status (SES) as a prospective predictor of log (forced expiratory volume in one second $[FEV]_1$/height$^2$) across young adulthood

| | Men | | Women | |
|---|---|---|---|---|
| **Effect on log ($FEV_1$/height$^2$)** | Model 1 [a] *b* (SE) | Model 2 [b] *b* (SE) | Model 1 [c] *b* (SE) | Model 2 [d] *b* (SE) |
| Initial pulmonary status (l/cm$^2$) | −8.96 (4.52E−3)*** | −8.96 (4.50E−3)*** | −9.09 (4.01E−3)*** | −9.10 (4.00E−3)*** |
| Childhood SES | 0.043 (4.67E−3)*** | 0.028 (4.97E−3)*** | 0.033 (3.85E−3)*** | 0.020 (4.10E−3)*** |
| Time from baseline (years) | −3.37E−3 (3.36E−4)*** | −3.34E−3 (3.41E−4)*** | −1.36E−3 (3.30E−4)*** | −1.34E−3 (3.33E−4)*** |
| Childhood SES × time | 1.58E−3 (5.13E−4)** | 1.59E−3 (5.21E−4)** | 1.89E−3 (4.76E−4)*** | 1.93E−3 (4.80E−4)*** |
| Age (years) | −5.10E−4 (8.87E−4) | −2.89E−3 (9.43E−4)** | −1.57E−3 (7.66E−4)* | −3.55E−3 (7.93E−4)*** |
| Age$^2$ (years$^2$) | −7.36E−4 (2.58E−4)** | −4.16E−4 (2.58E−4) | −7.06E−4 (2.22E−4)** | −4.49E−4 (2.20E−4)* |
| Current SES | — | 0.012 (01.99E−3)*** | — | 9.13E−3 (1.77E−3)*** |
| Asthma, unconfirmed [e] | — | −0.031 (0.015)* | — | −0.042 (0.011)*** |
| Asthma, confirmed by a doctor | — | −0.037 (4.51E−3)*** | — | −0.037 (4.02E−3)*** |
| Maternal smoking | — | 5.74E−3 (2.78E−3)* | — | −2.93E−3 (2.43E−3) |
| Paternal smoking | | −3.05E−3 (1.66E−3) | | −4.21E−3 (1.32E−3)** |
| Former smoker | — | 0.032 (9.47E−3)*** | — | 0.042 (7.84E−3)*** |
| Current smoker | — | 2.26E−3 (7.32E−3) | — | −2.96E−3 (6.34E−3) |

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

Note. Model 1 includes initial pulmonary status, adjusted for height$^2$; childhood SES; time from baseline; childhood SES × time interaction; age; and age$^2$. Model 2 includes all the variables in Model 1, as well as current SES; asthma, unconfirmed; asthma, confirmed by a doctor; maternal smoking; paternal smoking; participant's current smoking status; and participant's former smoking status. Total number of observations: for men = 6984; for women = 8355.

[a] 1157 observations are excluded because of missing values.

[b] 1262 observations are excluded because of missing values.

[c] 1425 observations are excluded because of missing values.

[d] 1523 observations are excluded because of missing values.

[e] Unconfirmed asthma was ascertained by answering yes to wheezing 'occasionally apart from colds' or 'most days or nights,' and to endorsing breathlessness 'when hurrying on the level of walking up a slight hill.'

participants with higher levels of childhood SES had higher levels of FVC (Table 3) among both men ($b = 0.052$, SE = 4.45E−3, $P < 0.001$) and women ($b = 0.038$, SE = 3.81E−3, $P = < = 0.001$). We observed an age$^2$ effect for both men and women $FEV_1$ and FVC), such that with a larger age$^2$ there was lower pulmonary function.

Using an interaction term of childhood SES and time in Model 1, we examined whether pulmonary function declined faster among participants with low versus higher childhood SES. A significant interaction term for men ($b = 1.58E-3$, SE = 5.13E−3, $P < 0.01$) and women ($b = 1.89E-3$, SE = 4.76E−3, $P < 0.001$) and subsequent examination of the associations suggested that $FEV_1$ was decreasing most rapidly among those with the lowest childhood SES. For men there was a significant decrease in $FEV_1$ at each level of childhood SES; those with the lowest childhood SES showed the most rapid decrease over the study period. For women there was a significant decrease in $FEV_1$ for the two lower childhood SES levels, but no change over time among those with the highest level. Similarly, a significant interaction term for FVC was seen for men ($b = 1.38E-3$, SE = 4.22E−4, $P < 0.01$) and women ($b = 1.31E-3$, SE = 3.94E−4, $P < 0.001$). Again, FVC was decreasing most rapidly among those with the lowest childhood SES. For men there was a significant decrease in FVC among those with the lowest childhood SES, whereas there was no change over time for those with higher childhood SES. For women there was no significant change in FVC for those with the lowest childhood SES, but there was significant increased growth among those with higher childhood SES.

Model 2—further adjusted for current SES, asthma history, and smoking history (parental and participant's own)—suggested that the independent effect of childhood SES remained for both men ($b = 0.028$, SE = 4.97E−3, $P < 0.001$) and women ($b = 0.020$, SE = 4.10E−3, $P < 0.001$). Similarly, participants with higher levels of childhood SES had higher levels of FVC (Table 3), as evidenced by a significant main effect of childhood SES for both men ($b = 0.039$, SE = 4.78E−3, $P < 0.001$) and women ($b = 0.028$, SE = 4.10E−3, $P < 0.001$). We again observed an age$^2$ effect for both men (with FVC) and women (with $FEV_1$), such that with a larger age$^2$ there was lower pulmonary function.

The interaction term for childhood SES and time in the fully adjusted Model 2 was consistent with the patterns in Model 1. Again, there was a significant interaction term for men ($b = 1.59E-3$, SE = 5.21E−4, $P < 0.01$) and women ($b = 1.93E-3$, SE = 4.80E−3, $P < 0.001$) and subsequent examination of the associations suggested that $FEV_1$ was decreasing most rapidly among those with the lowest childhood SES (Figures 1a and 1b). For men there was a significant decrease in $FEV_1$ at each level of childhood SES; those with the lowest childhood SES showed the most rapid decrease over the study period. Similarly, a significant interaction term for men ($b = 1.36E-3$, SE = 4.29E−4, $P < 0.01$) and women ($b = 1.31E-3$, SE = 3.96E−4, $P < 0.001$) and subsequent examination of the

**Table 3** Childhood socioeconomic status (SES) as a prospective predictor of log (forced vital capacity [FVC]/height$^2$) across young adulthood

| | Men | | Women | |
|---|---|---|---|---|
| **Effect on log (FVC/height$^2$)** | Model 1 [a] *b* (SE) | Model 2 [b] *b* (SE) | Model 1 [c] *b* (SE) | Model 2 [d] *b* (SE) |
| Initial pulmonary status (l/cm$^2$) | −8.73 (4.31E−3)*** | −8.74 (4.33E−3)*** | −8.91 (3.97E−3)*** | −8.92 (4.00E−3)*** |
| Childhood SES | 0.052 (4.45E−3)*** | 0.039 (4.78E−3)*** | 0.038 (3.81E−3)*** | 0.028 (4.10E−3)*** |
| Time from baseline (years) | −5.90E−4 (2.77E−4)* | −5.60E−4 (2.80E−4)* | 1.94E−3 (2.72E−4)*** | 1.96E−3 (2.74E−4)*** |
| Childhood SES × time | 1.38E−3 (4.22E−4)** | 1.36E−3 (4.29E−4)** | 1.31E−3 (3.94E−4)*** | 1.31E−3 (3.96E−4)*** |
| Age (years) | 2.56E−3 (8.46E−4)** | 4.53E−4 (9.08E−4) | 2.30E−3 (7.59E−4)** | 3.68E−4 (7.93E−4) |
| Age$^2$ (years$^2$) | −1.04E−3 (2.46E−4)*** | −7.50E−4 (2.49E−4)** | −5.70E−4 (2.20E−4)** | −2.28E−4 (2.21E−4) |
| Current SES | — | 0.011 (01.91E−3)*** | — | 8.47E−3 (1.78E−3)*** |
| Asthma, unconfirmed [e] | — | −0.019 (0.015) | — | −0.026 (0.011)* |
| Asthma, confirmed by a doctor | — | −0.017 (4.34E−3)*** | — | −0.025 (4.03E−3)*** |
| Maternal smoking | — | 8.55E−3 (2.68E−3)** | — | −1.51E−3 (2.43E−3) |
| Paternal smoking | | −3.28E−3 (1.60E−3)* | | −4.28E−3 (1.32E−3)** |
| Former smoker | — | 0.038 (9.12E−3)*** | — | 0.022 (7.85E−3)*** |
| Current smoker | — | 0.011 (7.05E−3) | — | 0.16 (6.35E−3)* |

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

Note. Model 1 includes initial pulmonary status, adjusted for height$^2$; childhood SES; time from baseline; childhood SES × time interaction; age; and age$^2$. Model 2 includes all the variables in Model 1, as well as current SES; asthma, unconfirmed; asthma, confirmed by a doctor; maternal smoking; paternal smoking; participant's current smoking status; and participant's former smoking status. Total number of observations: for men = 6984; for women = 8355.

[a] 1157 observations are excluded because of missing values.

[b] 1262 observations are excluded because of missing values.

[c] 1425 observations are excluded because of missing values.

[d] 1523 observations are excluded because of missing values.

[e] Unconfirmed asthma was ascertained by answering yes to wheezing 'occasionally apart from colds' or 'most days or nights,' and to endorsing breathlessness 'when hurrying on the level of walking up a slight hill.'

associations suggested that FVC was decreasing most rapidly among those with the lowest childhood SES (Figures 2a and 2b). For men there was a significant decrease in FVC among those with the lowest childhood SES, whereas there was no change over time for those with higher childhood SES. For women there was no significant change in FVC for those with the lowest childhood SES, but there was actually significant increased growth among those with higher childhood SES.

Derived from Model 2, Figures 1 and 2 illustrate changes in young adult pulmonary function for both men and women over the 5-year study period, as a function of childhood SES. Projecting these differential effects 15 years beyond baseline, we calculated the relative change in pulmonary function by comparing those with the lowest childhood SES to those with the highest childhood SES.[‡] Fifteen years from baseline men with the highest childhood SES showed a 0.5% decrease in $FEV_1$ (i.e. losing 165 cc), whereas men with the lowest childhood SES showed a 1.2% decrease (losing 421 cc); women with the highest childhood SES showed an 0.1% decrease in $FEV_1$ (losing 4 cc), whereas women with the lowest childhood SES showed a 0.8% decrease (losing 187 cc). For FVC, 15 years from baseline men with the highest childhood SES showed a 1% increase (gaining 7 cc), whereas men with the lowest childhood SES showed a 0.5% decrease (losing 199 cc); women with the highest childhood SES showed a 4.1% increase in FVC (gaining 152 cc) whereas women with the lowest childhood SES showed a 0.2% decrease (losing 32cc).

Some research suggests confounding or interactive effects between race/ethnicity and SES in relation to health outcomes.[24–26] However, little research to date has looked at these issues in relation to pulmonary function. Ideally to do so one would examine a childhood SES × gender × race interaction over the study period. However, in the current sample, this stratification leads to small cell sizes resulting in unstable estimates due to inadequate statistical power. However, in exploratory analyses we looked within racial groups, controlling for gender, to see if the childhood SES effect was present (data not shown). For all groups, trends suggested that lower SES compared with higher SES was associated with lower pulmonary function.

## Discussion

These prospective data are the first to link childhood SES with pulmonary function levels and change across young adulthood, and suggest that effects accumulate over time. Childhood SES predicted baseline pulmonary function, subsequent levels of pulmonary function, and rates of decline in young adult men and women, even adjusting for current SES, height, age, and age$^2$. A graded association between childhood SES and young adult pulmonary function was evident over the follow-up period. Maximally attained pulmonary function is purportedly achieved during young adulthood, and these data suggest that lower levels are linked to lower childhood SES, beyond the effects of current SES. This is important because maximally

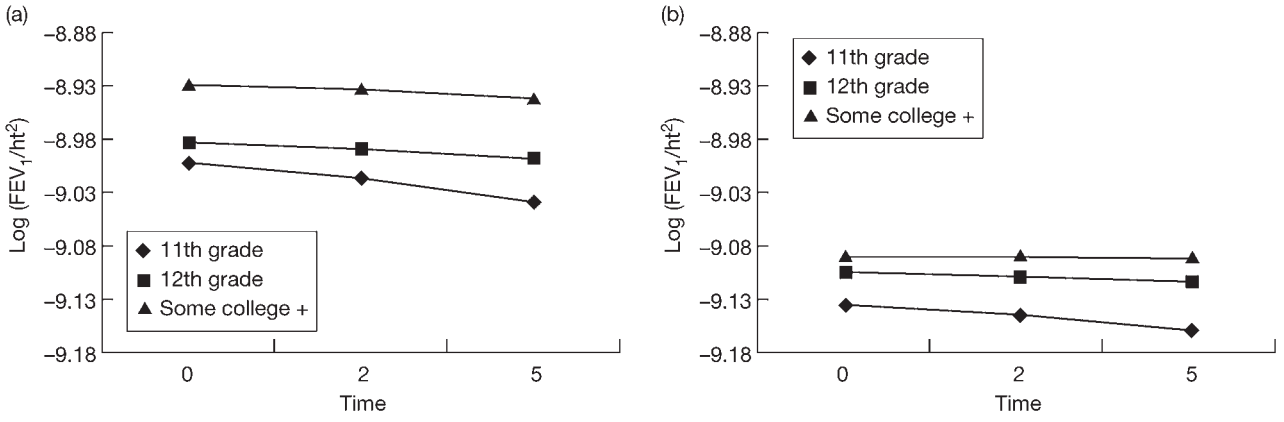


**Figure 1a** Log (forced expiratory volume in one second [$FEV_1$]/$ht^2$) by childhood socioeconomic status (SES) over time, men **b** Log (forced expiratory volume in one second [$FEV_1$]/$ht^2$) by childhood socioeconomic status (SES) over time, women

Note. Adjusted for initial pulmonary status, age, $age^2$, current SES, asthma (unconfirmed and confirmed), parental smoking status (maternal and paternal), participant smoking status.

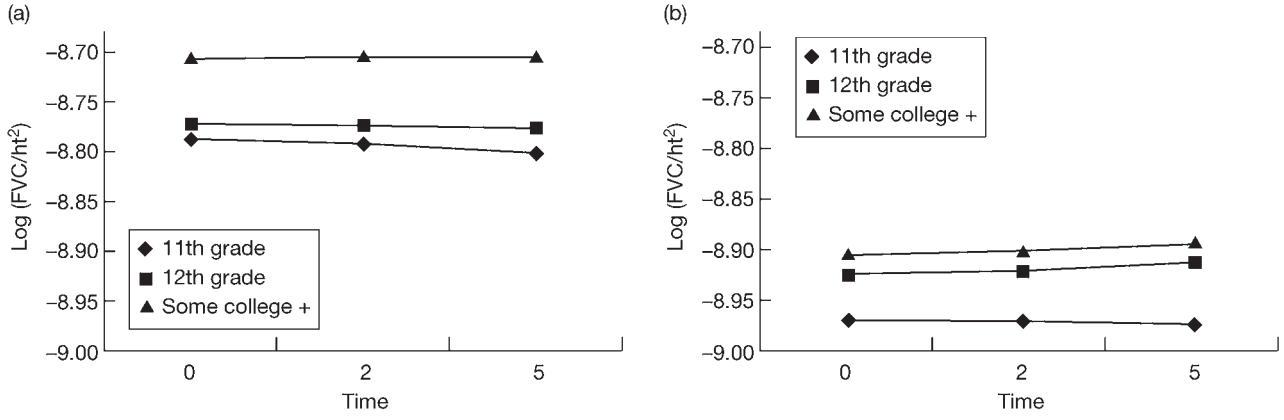


**Figure 2a** Log (forced vital capacity [FVC]/$ht^2$) by childhood socioeconomic status (SES) over time, men **b** Log (forced vital capacity [FVC]/$ht^2$) by childhood socioeconomic status (SES) over time, women

Note. Adjusted for initial pulmonary status, age, $age^2$, current SES, asthma (unconfirmed and confirmed), parental smoking status (maternal and paternal), participant smoking status.

attained pulmonary function may determine the buffer one has against developing COPD and other health problems in later life.[1] Moreover, when accounting for baseline pulmonary function, participants with lower childhood SES showed earlier onset of pulmonary function decline relative to those with higher childhood SES.

Previous work has suggested that the plateau phase of pulmonary development in young adulthood actually is not a steady-state period.[27] Indeed, our FVC findings for women (Figure 2b) suggest that pulmonary function was *increasing* in the highest while declining in the lowest childhood SES group. Replication of the trajectory we found may suggest underestimation of the length of the growth phase because of failure to consider pulmonary function in the context of SES, and the conditions (e.g. housing, workplace exposures), medical history (e.g. asthma), and behaviours (e.g. smoking) that are shaped by SES.

We included several covariates that may mediate the childhood SES—young adult pulmonary function relationship. Though we did not conduct formal tests of mediation, our analyses suggest that childhood SES may affect both history of asthma and smoking, which in turn may influence pulmonary function. Indeed, the addition of these terms (along with current SES) substantially reduced the effect on pulmonary function of childhood SES and the interaction of childhood SES with time (Tables 2 and 3), while themselves remaining significant. Notably, those with lower childhood SES were more likely to report *undiagnosed* asthma symptoms, but less likely to report *diagnosed* asthma. This may reflect the relationship between SES and both access to healthcare and the management and treatment of asthma.[28] In turn, asthma predicted decreases in $FEV_1$ and FVC for both men and women. The relationships among childhood SES, smoking, and pulmonary outcomes were more complicated and warrant

further investigation. Of key importance is that while asthma history and smoking may partially mediate the childhood SES—young adult pulmonary function link, an effect of childhood SES persists beyond the effects of these variables. Even accounting for the effects of asthma history and smoking, projections 15 years from baseline showed the effects of childhood SES on pulmonary function only more pronounced.

It is also noteworthy that after adjusting for all of these covariates, we observed an age$^2$ effect for both men (with FVC) and women (with $FEV_1$), such that with a larger age$^2$ there was lower pulmonary function. Due to different exposures occurring at different times in history, not only *that* one is 25 years old may influence pulmonary function (age effects), but also *when* one reaches a given age (cohort effects) is important to consider. Our findings suggest that adjusting for time from baseline (i.e. cohort effects) and other covariates, even across young adulthood in some cases pulmonary function declines more rapidly with age.

Other mechanisms may influence the relationship between childhood SES and young adult pulmonary function.[29] For example, children of low SES tend to live in environments exposing them to toxins ranging from air pollution (indoor and outdoor) to interpersonal violence.[30–32] These toxins may promote respiratory infection; directly, as with air pollution,[33] or indirectly through routes like stress, as with interpersonal violence.[34] Such exposure may in turn influence later-life pulmonary function.[19] Similarly, burgeoning scholarship suggests that psychological factors (e.g. negative emotions, optimism) may be another important route linking social structure and health.[35,36] Childhood SES may also shape health behaviours beyond smoking that affect later pulmonary health. For example, nutritional intake of foods high in antioxidants may be protective against pulmonary decline.[37]

We cannot fully rule out the possibility that there is an unexamined third factor leading to low childhood SES, low pulmonary function, and increased rates of pulmonary function decline. For example, poor health from inherited chronic conditions may influence parents' SES and participants' pulmonary function levels and decline. Though the prospective findings provide important evidence for causation in the direction of childhood SES influencing subsequent pulmonary function and change, we did not test reverse-causal hypotheses in the current study.

Our measure of childhood SES was admittedly limited and subject to recall bias. However, recall of parents' education may be less subject to memory bias than answering more specific questions about living conditions two decades or more earlier. In addition, participants were unaware of our interest in the link between childhood SES and pulmonary function, making a systematic bias in any one direction less likely. Given the restricted range of the childhood SES measure, its significant effect on young adult pulmonary function suggests a robust relationship.

We did not have the power to stratify by race and gender in order to understand childhood SES effects on pulmonary function among black women, white women, black men, and white men, but results were qualitatively similar across these groups. In future work it will be imperative to sample participants in a way to enable the examination of childhood SES $\times$ gender $\times$ race interactions.

These data add to the growing evidence of SES disparities in physical health. Young adulthood is an under-studied but developmentally critical phase for health because maximum level of pulmonary function is attained during this period, setting the stage for later-life resilience or rapid decline. Our findings suggest that a life-course approach may be usefully applied to pulmonary function in young adulthood. Childhood SES may influence young adulthood pulmonary function in two ways. First, individuals with lower childhood SES may not attain as high levels of pulmonary function in early adulthood relative to individuals with higher childhood SES. Second, pulmonary function may decline earlier and more quickly for individuals with lower childhood SES. Given that pulmonary function decline is progressive, future research is needed to explicate the implications of these findings for later-life health status, as well as the mechanisms linking childhood SES and young adult pulmonary function.

## Acknowledgements

These analyses were supported by the MacArthur Foundation Network on Socioeconomic Status and Health. During preparation of this manuscript B Jackson was supported by a training grant from the National Heart, Lung, and Blood Institute (HL07427). S Cohen was supported by a Senior Scientist Award from the National Institute of Mental Health (MH00721). S Weiss was supported by HL07427. RJ Wright was supported by K08 HL04187. The CARDIA study is conducted and supported by the NHLBI in collaboration with the CARDIA study investigators. This manuscript was not prepared in collaboration with investigators of the CARDIA study and does not necessarily reflect the opinions or views of the CARDIA study or the NHLBI.

**KEY MESSAGES**

- Childhood socioeconomic status (SES) may influence pulmonary function in young adulthood.
- Pulmonary function may show lower levels, and earlier, faster decline for individuals with relatively lower childhood SES.
- Effects of childhood SES on young adult pulmonary function are similar for men and women.
- These findings are important because pulmonary function in young adulthood may shape chronic obstructive pulmonary disease and other later-life health outcomes.

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