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

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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 centers 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 FEV1 (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 FEV1 and FVC differed in a gradient fashion by childhood SES. As shown by significant childhood SES by time interaction terms, these associations with FEV1 were robust for men \( (b = 1.59 \times 10^{-3}, \ SE = 5.21 \times 10^{-4}, \ P < 0.001) \) and women \( (b = 1.93 \times 10^{-3}, \ SE = 4.80 \times 10^{-4}, \ P < 0.001) \), and adjusted for multiple potential confounders including smoking. Results were similar for FVC. Subsequent examination of the interaction terms suggested that FEV1 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

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* 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. COPD, for instance, results in poor physical, psychological, and social functioning, poses a considerable economic burden, and is projected to be the third leading cause of death by 2020. Factors contributing to poor pulmonary function have not yet been elucidated fully.

Researchers have conceptualized socioeconomic factors as a ‘fundamental cause’ of disease. However, few studies go beyond considering socioeconomic status (SES) as a confounder when examining pulmonary function. 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. 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. Because childhood SES may be linked with differential exposure to a variety of risk factors (e.g. asthma, allergens, tobacco smoke) resulting in childhood respiratory trouble, childhood SES may be a critical predictor of adult pulmonary function. Young adults are important to study because maximally attained pulmonary function is achieved in young adulthood, 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, 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.

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 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. 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 for men. 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. This allows examination of how people change over time, and has been used to examine pulmonary change. 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. 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 FEV1 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. 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). 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 age2. 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 FEV1 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 FEV1 (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,
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

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### 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

<table>
<thead>
<tr>
<th>Effect on log (FEV(_1)/height(^2))</th>
<th>Men Model 1 (^a)</th>
<th>Men Model 2 (^b)</th>
<th>Women Model 1 (^c)</th>
<th>Women Model 2 (^d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial pulmonary status (l/cm(^2))</td>
<td>(-8.96 (4.52E-3)**)</td>
<td>(-8.96 (4.50E-3)**)</td>
<td>(-9.09 (4.01E-3)**)</td>
<td>(-9.10 (4.00E-3)**)</td>
</tr>
<tr>
<td>Childhood SES</td>
<td>(0.043 (4.67E-3)**)</td>
<td>(0.028 (4.97E-3)**)</td>
<td>(0.033 (3.85E-3)**)</td>
<td>(0.020 (4.10E-3)**)</td>
</tr>
<tr>
<td>Time from baseline (years)</td>
<td>(-3.37E-3 (3.36E-4)**)</td>
<td>(-3.34E-3 (3.41E-4)**)</td>
<td>(-1.36E-3 (3.30E-4)**)</td>
<td>(-1.34E-3 (3.33E-4)**)</td>
</tr>
<tr>
<td>Childhood SES (\times) time</td>
<td>(1.58E-3 (5.13E-4)**)</td>
<td>(1.59E-3 (5.21E-4)**)</td>
<td>(1.89E-3 (4.76E-4)**)</td>
<td>(1.93E-3 (4.80E-4)**)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>(-5.10E-4 (8.87E-4)**)</td>
<td>(-2.89E-3 (9.43E-4)**)</td>
<td>(-1.57E-3 (7.66E-4)**)</td>
<td>(-3.55E-3 (7.93E-4)**)</td>
</tr>
<tr>
<td>Age(^2) (years(^2))</td>
<td>(-7.36E-4 (2.58E-4)**)</td>
<td>(-4.16E-4 (2.58E-4)**)</td>
<td>(-7.06E-4 (2.22E-4)**)</td>
<td>(-4.49E-4 (2.20E-4)**)</td>
</tr>
<tr>
<td>Current SES</td>
<td>(-0.012 (0.99E-3)**)</td>
<td>(-0.031 (0.015)*)</td>
<td>(-0.042 (0.011)**)</td>
<td>(9.13E-3 (1.77E-3)**)</td>
</tr>
<tr>
<td>Asthma, unconfirmed (^e)</td>
<td>(-0.037 (4.51E-3)**)</td>
<td>(-0.037 (4.02E-3)**)</td>
<td>(-0.037 (4.02E-3)**)</td>
<td>(-0.037 (4.02E-3)**)</td>
</tr>
<tr>
<td>Asthma, confirmed by a doctor</td>
<td>(5.74E-3 (2.78E-3)**)</td>
<td>(-2.93E-3 (2.43E-3)**)</td>
<td>(-2.93E-3 (2.43E-3)**)</td>
<td>(-2.93E-3 (2.43E-3)**)</td>
</tr>
<tr>
<td>Maternal smoking</td>
<td>(-3.05E-3 (1.66E-3)**)</td>
<td>(-4.21E-3 (1.32E-3)**)</td>
<td>(-4.21E-3 (1.32E-3)**)</td>
<td>(-4.21E-3 (1.32E-3)**)</td>
</tr>
<tr>
<td>Paternal smoking</td>
<td>(-0.032 (9.47E-3)**)</td>
<td>(-0.042 (7.84E-3)**)</td>
<td>(-0.042 (7.84E-3)**)</td>
<td>(-0.042 (7.84E-3)**)</td>
</tr>
<tr>
<td>Former smoker</td>
<td>(-2.26E-3 (7.32E-3)**)</td>
<td>(-2.96E-3 (6.34E-3)**)</td>
<td>(-2.96E-3 (6.34E-3)**)</td>
<td>(-2.96E-3 (6.34E-3)**)</td>
</tr>
</tbody>
</table>

\(^a\) \(1157\) 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.’

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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 higher childhood SES. For FVC, 15 years from baseline men with the highest 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).

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 age². 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
attained pulmonary function may determine the buffer one has against developing COPD and other health problems in later life. 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. Indeed, our FVC findings for women 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. In turn, asthma predicted decreases in FEV₁ and FVC for both men and women. The relationships among childhood SES, smoking, and pulmonary outcomes were more complicated and warrant further investigation.

**Figure 1a** Log (forced expiratory volume in one second [FEV₁]/ht²) by childhood socioeconomic status (SES) over time, men

**Figure 1b** Log (forced expiratory volume in one second [FEV₁]/ht²) by childhood socioeconomic status (SES) over time, women

Note. Adjusted for initial pulmonary status, age, age², current SES, asthma (unconfirmed and confirmed), parental smoking status (maternal and paternal), participant smoking status.

**Figure 2a** Log (forced vital capacity [FVC]/ht²) by childhood socioeconomic status (SES) over time, men

**Figure 2b** Log (forced vital capacity [FVC]/ht²) by childhood socioeconomic status (SES) over time, women

Note. Adjusted for initial pulmonary status, age, age², current SES, asthma (unconfirmed and confirmed), parental smoking status (maternal and paternal), participant smoking status.
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 FEV1), 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 × gender × 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.

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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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