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The Potential Effects of Policy-driven Air Pollution Interventions on Childhood Lung Development

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Abstract

Rationale: Although elevated air pollution exposure impairs lung-function development in childhood, it remains a challenge to use this information to estimate the potential public health benefits of air pollution interventions in exposed populations.

Objectives: Apply G-computation to estimate hypothetical effects of several realistic scenarios for future air pollution reductions on lung growth.

Methods: Mixed-effects linear regression was used to estimate FEV_1 and FVC from age 11 to 15 years in 2,120 adolescents across 3 cohorts (1993–2001, 1997–2004, and 2007–2011). Models included regional pollutants (nitrogen dioxide [NO_2] or particulate matter with an aerodynamic diameter $\leq$2.5 μm [$PM_{2.5}$]) and other important covariates. Using G-computation, a causal inference–based method, we then estimated changes in mean lung growth in our population for hypothetical interventions on either NO_2 or $PM_{2.5}$. Confidence intervals (CIs) were computed by bootstrapping ($N = 1{,}000$).

Measurements and Main Results: Compared with the effects of exposure from observed NO_2 concentrations during the study period, had communities remained at 1994 to 1997 NO_2 levels, FEV_1 and FVC growth were estimated to have been reduced by 2.7% (95% CI, −3.6 to −1.8) and 4.2% (95% CI, −5.2 to −3.4), respectively. If NO_2 concentrations had been reduced by 30%, we estimated a 4.4% increase in FEV_1 growth (95% CI, 2.8–5.9) and a 7.1% increase in FVC growth (95% CI, 5.7–8.6). Comparable results were observed for $PM_{2.5}$ interventions.

Conclusions: We estimated that substantial increases in lung function would occur as a result of interventions that reduce NO_2 or $PM_{2.5}$ concentrations. These findings provide a quantification of potential health benefits of air quality improvement.

Keywords: lung function; air pollution; children; nitrogen dioxide; particulate matter

Adults with reduced lung function are at increased risk of chronic obstructive pulmonary disease (COPD), cardiovascular disease, and all-cause mortality (1–4). There is evidence that low lung function in early life predisposes individuals to having low lung function throughout life (5), and studies have shown low lung function in early adulthood to be associated with subsequent development of disease, including COPD (6). There is general consensus that some respiratory diseases occurring in adulthood have their origins in early life (7–9).

Many studies have shown that air pollution is associated with reduced lung-function levels, especially in children (10–12). In the Southern California Children's Health Study (CHS), several regional air pollutants (e.g., general levels of air pollutants in a community) were associated with slower lung-function growth and reduced attained level by the end of childhood (13, 14). In a recent study from the CHS, improvements in lung function were observed with declining levels of nitrogen dioxide (NO_2) and

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At a Glance Commentary

Scientific Knowledge on the Subject: Exposure to air pollution has been shown to impair lung-function development. It has also been shown that improving air quality leads to better lung function, but estimating the potential public health benefits of different air pollution interventions is challenging.

What This Study Adds to the Field: We estimated increases in lung function under several scenarios of reduced nitrogen dioxide or concentrations of particulate matter with an aerodynamic diameter $\leq$2.5 μm, some of which were informed by existing air pollution standards. This is important because improving lung function in childhood could have long-term positive impacts on public health.

particulate matter with an aerodynamic diameter $\leq$2.5 μm ($PM_{2.5}$) in school-aged children across three cohorts spanning two decades, a period during which there were large decreases in concentrations of both these pollutants (15). Significant associations of declining exposure with improved lung-function growth were observed in communities with NO_2 concentrations well below the U.S. Environmental Protection Agency (EPA) annual standard of 53 ppb. This finding suggests the existing EPA annual NO_2 standard may not be sufficient to protect human health.

Although this study and others have shown large and statistically significant associations between air pollution levels and lung function (10–15), because effect estimates are conditional on model covariates, use of estimates from these studies do not adequately reflect what would happen to public health if there were an intervention to shift community exposures; for example, what would be the net improvement in lung function if exposure to regional pollutants were lowered to specified concentrations in a subset or in all participants compared with what was observed. G-computation, a method used for causal inference, is one approach that can be used to estimate and compare how shifts in exposures under different intervention scenarios may result in shifts in the outcome distribution (16–18). That is, G-computation allows for the estimation of marginal, rather than conditional, mean lung-function growth by computing a standardized mean outcome across the observed covariate distribution (e.g., mean lung-function growth between ages 11 and 15 years across the sample); exposure effects are then estimated by contrasting standardized mean outcomes under different exposure scenarios.

The current study expands upon prior findings in these cohorts (15) by using G-computation to compare lung-function development under different hypothetical air pollution exposure scenarios relative to the observed exposures in our data. This study focuses on scenarios involving interventions on NO_2 and $PM_{2.5}$ concentrations because these two pollutants showed the strongest associations with lung function in the earlier analysis. Some of the results of this study have been previously reported in the form of an abstract (19).

Methods

Participants

Details of the study design have been described elsewhere (15). Briefly, the study sample included 2,120 children from five study communities (Long Beach, Mira Loma, Riverside, San Dimas, and Upland) that were recruited across three separate cohorts. The first two cohorts recruited students from fourth-grade classrooms in 1993 (1993 cohort, $n = 669$) and 1996 (1996 cohort, $n = 588$), and the third cohort recruited students from kindergarten or first-grade classrooms in 2002 (2002 cohort, $n = 863$). Key covariate information pertaining to participants, including sex, race or ethnicity, age, parental education, asthma status, and respiratory-tract illness on the lung-function test day, was obtained from questionnaires completed by either parents or participants.

Lung-Function Testing

Trained field technicians measured FEV_1, FVC, height, and weight during lung-function testing visits. In the 1993 and 1996 cohorts, lung function was measured every year from grade 4 to grade 12 (ages 10–18 yr) using rolling-seal spirometers; in the 2002 cohort, lung function was measured every other year when children were approximately 11, 13, and 15 years of age using pressure transducer-based spirometers. Lung growth from 11 to 15 years of age was the primary outcome of interest because lung-function measurements were made at these ages in all three cohorts.

Air Pollutants

Central site monitors have been continuously measuring criteria pollutants in all study communities since 1994. Four-year mean concentrations were computed for NO_2 and $PM_{2.5}$ by community for each cohort, corresponding to lung-function growth measurements from 11 to 15 years of age (1994–1997 for the 1993 cohort, 1997–2000 for the 1996 cohort, and 2007–2010 for the 2002 cohort).

Statistical Analysis

G-computation is a statistical method that aims to make causal inferences by estimating unbiased marginalized effects from observational data based in the counterfactual framework (i.e., there are many potential outcomes given all possible exposures, of which we only observed one) (17, 18, 20). Also referred to as a substitution estimator, G-computation can be used to estimate shifts in outcome within a population as a result of shifts in exposure (16). This allows an estimated answer to the question, "What would have been the observed health outcomes in a population had their exposure been different than what was actually experienced?" This is a causal question planted in the counterfactual framework (17, 18, 20). G-computation was used to estimate lung-function growth from ages 11 to 15 years if the three different cohorts had experienced different levels of exposure to air pollutants based on several hypothetical scenarios, which was then contrasted to lung growth based on observed exposures.

G-computation was implemented in a series of steps. In step 1, a mixed effects linear regression model was fitted using observed data (i.e., observed pollutant exposures, covariate information, and all available lung-function measurements) to obtain the relationship between lung function and predictors. Specifically, we used mixed-effects linear regression model developed in our previous studies (13, 15), which included air pollutant exposure, sex, race, Hispanic ethnic background, age (with

knots placed at ages 12, 14, and 16 years to allow for nonlinear growth of lung function during adolescence), height, height squared, body mass index, body mass index squared, respiratory-tract illness on the lung-function test day, and study community, as well as two-way interactions of age with air pollutant exposure, sex, race, and Hispanic ethnic background. The model additionally included an individual-level random intercept to account for repeated measures of lung function by an individual. Unlike previously used models, a random effect to account for clustering of individuals at the community-cohort level was not included in the model. We noted effect estimates were not markedly different between models including and excluding this additional random effect parameter, thus we excluded this parameter for computational efficiency. Calculations of 95% confidence intervals (CIs) were not based on model-derived SEs but instead by using the bootstrap method, as noted below. In step 2, lung function was predicted at all ages for each individual under different exposure scenarios by using the model specified in step 1 and altering air pollutant concentration; covariate values were not altered. In step 3, predicted lung function was regressed against age, with knots at ages 12, 14, and 16 years, to obtain estimates of lung growth from age 11 to 15 years for each exposure scenario. In step 4, estimates of lung-function growth under each exposure scenario were than contrasted to lung-function growth under observed exposures. CIs were estimated by bootstrapping the data with replacement 1,000 times (21), repeating steps 1 to 4 as outlined, and reporting the 2.5 and 97.5 percentiles from the distribution of estimates. Estimated differences in mean lung function were based on the mean outcomes among the entire study population and not restricted only to individuals for whom exposure assignments were altered.

Lung growth based on several hypothetical air pollution scenarios were considered in addition to one based on observed exposures. Interventions on single pollutants were studied because these most closely align with air quality standards, which are set for one pollutant at a time (e.g., EPA National Ambient Air Quality Standards). Moreover, high correlation between regional NO_2 and $PM_{2.5}$ in our data precluded inclusion of both pollutants in a single model. The first set of scenarios examined the effect on lung function had NO_2 or $PM_{2.5}$ concentrations remained the same as in the 1993 cohort (i.e., had the air pollution declines observed during the two decades of the CHS not happened). In this scenario, all participants in the 1996 and 2002 cohorts were exposed to the same community-specific concentrations of NO_2 or $PM_{2.5}$ as participants in the 1993 cohort. This first scenario quantified the public health benefit of actual observed air quality improvements. The remaining air pollution intervention scenarios were selected to demonstrate the impact on lung-function growth had air pollutant concentration been lower than what was observed. The second set of air pollution scenarios examined the effect on lung function had all participants across the three cohorts been exposed to NO_2 or $PM_{2.5}$ concentrations that were 10%, 20%, or 30% lower than actually observed. These percentage-based scenarios, rather than absolute reductions, better follow a more typical pattern of air quality improvements whereby communities with higher air pollution levels have greater absolute reductions in air pollution compared with lower air pollution communities. Furthermore, some have argued for air pollution regulations based on rates of change, rather than compliance to a standard (22), which this set of air pollution scenarios emulates. The last set of air pollution scenarios examined the effect on lung function had communities not exceeded specific thresholds for NO_2 or $PM_{2.5}$, specifically 30 and 20 ppb for NO_2, and 15 and 12 $\mu g/m^3$ for $PM_{2.5}$. In this scenario, in which exposures were set equal to the threshold, only individuals in communities above the threshold had their exposures changed. This last set of scenarios simulates strict adherence to hypothetical air quality standards. These thresholds were selected after considering the current NO_2 and $PM_{2.5}$ standards set by the EPA (53 ppb for NO_2 and 12 $\mu g/m^3$ for $PM_{2.5}$) (23) and the World Health Organization (21.3 ppb for NO_2 and 10 $\mu g/m^3$for $PM_{2.5}$) (24). We also examined the observed air pollutants distribution and noted no communities exceeded 40 ppb NO_2 or were below 10 $\mu g/m^3$ $PM_{2.5}$. The three sets of air pollution scenarios are depicted in Tables E1 and E2 in the online supplement.

Results

Details of this study population have been previously reported (15) and are partially reproduced in Table 1. Among the 2,120 study participants, there were slightly fewer males (48%) than females (52%) with no significant difference in sex distribution by cohort ($P = 0.49$; *see* Table 1). There was an increase in the number of participants of Hispanic ethnicity from the 1993 cohort (31.0%) to the 2002 cohort (57.6%; $P < 0.001$). NO_2 and $PM_{2.5}$ concentrations were highest in the 1993 cohort (median NO_2, 34.4 ppb; median $PM_{2.5}$, 28.7 $\mu g/m^3$) and lowest in the 2002 cohort (median NO_2, 21.4 ppb; median $PM_{2.5}$, 13.0 $\mu g/m^3$). The correlation coefficient for changes in community-specific mean NO_2 and $PM_{2.5}$ concentrations from 1994–1997 to 2007–2010 was 0.82 (15). Table E3 contains the number of community-cohort combinations that were above each threshold for the examined threshold scenarios.

We estimated that substantial improvements in lung development have occurred from past interventions on air pollution and that there are large potential benefits from a range of possible community-level interventions. In the observed data, 4-year FEV_1 growth from age 11 to 15 years was 1,183.8 ml among all individuals across all cohorts and communities (Table 2). Had NO_2 concentrations remained at levels observed in the 1993 cohort, the earliest time period with highest air pollution of all three cohorts, we would have estimated this 4-year FEV_1 growth to be 2.7% smaller (95% CI, −3.6 to −1.8) compared with observed growth. However, in a scenario in which NO_2 concentrations had been reduced by 30%, we estimated FEV_1 growth would be 4.4% larger than what was observed (95% CI, 2.8–5.9). If communities had not exceeded a threshold of 30 ppb for NO_2, we estimated a modest 1% increase (95% CI, 0.6–1.4) in FEV_1 growth, and if communities had not exceeded a stricter threshold of 20 ppb for NO_2, we estimated a 4% increase (95% CI, 2.5–5.4) in FEV_1 growth compared with observed growth.

Substantial benefits to FEV_1 growth were similarly estimated as a result of

Table 1. Demographic Characteristics

Variable	All (*N* = *2,120*)	Cohort C (*n* = *669*)	Cohort D (*n* = *588*)	Cohort E (*n* = *863*)	*P* Value*
Sex, M, *n* (%)					
No	1,105 (52.1)	344 (51.4)	298 (50.7)	463 (53.7)	0.49
Yes	1,015 (47.9)	325 (48.6)	290 (49.3)	400 (46.3)	
Race, *n* (%)					
Asian	157 (7.8)	61 (9.4)	47 (8.0)	49 (6.3)	<0.001
Black	126 (6.3)	60 (9.2)	43 (7.4)	23 (3.0)	
Mixed	229 (11.4)	47 (7.2)	57 (9.7)	125 (16.1)	
Other	469 (23.4)	130 (20.0)	115 (19.7)	224 (28.9)	
White	1,027 (51.1)	351 (54.1)	323 (55.2)	353 (45.6)	
Hispanic ethnicity, *n* (%)					
Yes	870 (42.1)	204 (31.0)	193 (32.9)	473 (57.6)	<0.001
No	1,196 (57.9)	455 (69.0)	393 (67.1)	348 (42.4)	
Asthma at baseline, *n* (%)					
No	1,646 (82.2)	536 (86.3)	465 (82.9)	645 (78.7)	<0.001
Yes	356 (17.8)	85 (13.7)	96 (17.1)	175 (21.3)	
Parental education, *n* (%)					
Did not finish high school	300 (14.8)	107 (16.5)	61 (10.7)	132 (16.3)	0.0105
High school diploma or some college	1,167 (57.6)	372 (57.4)	352 (62.0)	443 (54.6)	
College diploma or greater	560 (27.6)	169 (26.1)	155 (27.3)	236 (29.1)	

**P* value calculated using the chi-squared test.

lowering $PM_{2.5}$ levels (*see* Table 2). For example, we estimated a reduction of $PM_{2.5}$ by 30% would have resulted in a 2.5% increase in FEV_1 growth (95% CI, 1.4–3.9) compared with observed growth. Additionally, we found FEV_1 growth to be higher if communities with high $PM_{2.5}$ levels were lowered to different thresholds. For example, if communities did not exceed a threshold of 15 $\mu g/m^3$, FEV_1 growth was estimated to be 2.6% higher (95% CI, 1.4–4.0) and, for a threshold of 12 $\mu g/m^3$, it would be 3.6% larger (95% CI, 2.0–5.6). Similar benefits were estimated for FVC growth for each scenario in which pollutant concentrations were lowered (Table 3).

Discussion

Although previous studies have shown that there are adverse effects of air pollution on lung development, this study quantified differences in lung-function growth under several scenarios of changing air quality from hypothetical community interventions.

Table 2. Estimates of the Effect of Hypothetical Air Pollutant Interventions on FEV_1 Growth between Ages 11 and 15 Years

	Mean (95% CI) FEV_1 Growth from Ages 11 to 15 yr (*ml*)	Absolute Change (95% CI)	Percent Change (95% CI)
NO_2			
Observed exposure*	1,183.8 (1,164.5 to 1,199.2)	Reference	Reference
Remain at cohort C levels†	1,152.1 (1,129.7 to 1,169.2)	−31.7 (−42.6 to −20.9)	−2.7 (−3.6 to −1.8)
Percent reduction			
10%	1,201.0 (1,181.0 to 1,218.0)	17.2 (11.2 to 23.4)	1.5 (0.9 to 2.0)
20%	1,218.2 (1,194.6 to 1,238.8)	34.4 (22.4 to 46.8)	2.9 (1.9 to 4.0)
30%	1,235.4 (1,208.3 to 1,261.0)	51.6 (33.4 to 70.2)	4.4 (2.8 to 5.9)
Hypothetical thresholds‡			
30 ppb	1,195.8 (1,176.2 to 1,212.3)	12.0 (7.7 to 16.8)	1.0 (0.6 to 1.4)
20 ppb	1,229.7 (1,203.6 to 1,253.6)	45.9 (29.7 to 63.3)	3.9 (2.5 to 5.4)
$PM_{2.5}$			
Observed exposure*	1,182.8 (1,163.8 to 1,198.4)	Reference	Reference
Remain at cohort C levels†	1,152.2 (1,127.9 to 1,170.9)	−30.6 (−46.0 to −17.1)	−2.6 (−3.9 to −1.5)
Percent reduction			
10%	1,192.8 (1,146.1 to 1,183.0)	10.0 (5.5 to 15.4)	0.8 (0.5 to 1.3)
20%	1,202.9 (1,160.5 to 1,196.0)	20.1 (10.8 to 30.7)	1.7 (0.9 to 2.6)
30%	1,212.9 (1,174.1 to 1,209.9)	30.1 (16.2 to 46.1)	2.5 (1.4 to 3.9)
Hypothetical thresholds‡			
15 $\mu g/m^3$	1,213.6 (1,190.0 to 1,236.6)	30.8 (16.4 to 47.7)	2.6 (1.4 to 4.0)
12 $\mu g/m^3$	1,225.5 (1,199.6 to 1,253.2)	42.7 (23.0 to 65.8)	3.6 (2.0 to 5.6)

Definition of abbreviations: CI = confidence interval; NO_2 = nitrogen dioxide; $PM_{2.5}$ = particulate matter with an aerodynamic diameter ≤2.5 μm.
*Predicted outcome based on observed exposure.
†Predicted outcome if all participants in cohorts D and E had experienced exposures equal to that of participants in cohort C.
‡Predicted outcome if all participants in communities that exceeded each threshold had been assigned exposures equal to that threshold.

Table 3. Estimates of the Effect of Hypothetical Air Pollutant Interventions on FVC Growth between Ages 11 and 15 Years

	Mean (95% CI) FVC Growth from Ages 11 to 15 yr (*ml*)	Absolute Change (95% CI)	Percent Change (95% CI)
NO_2			
Observed exposure*	1,368.7 (1,347.7 to 1,387.5)	Reference	Reference
Remain at cohort C levels†	1,311.2 (1,287.0 to 1,330.0)	−57.5 (−71.0 to −46.1)	−4.2 (−5.2 to −3.4)
Percent reduction			
10%	1,401.0 (1,378.9 to 1,422.7)	32.3 (25.8 to 39.5)	2.4 (1.9 to 2.9)
20%	1,433.3 (1,408.2 to 1,459.1)	64.6 (51.7 to 79.0)	4.7 (3.8 to 5.8)
30%	1,465.6 (1,435.2 to 1,496.9)	96.9 (77.5 to 118.5)	7.1 (5.7 to 8.6)
Hypothetical thresholds‡			
30 ppb	1,391.7 (1,369.9 to 1,412.5)	23.0 (18.1 to 28.1)	1.7 (1.3 to 2.1)
20 ppb	1,455.9 (1,426.6 to 1,485.4)	87.2 (69.1 to 106.5)	6.4 (5.0 to 7.8)
$PM_{2.5}$			
Observed exposure*	1,367.9 (1,347.4 to 1,386.9)	Reference	Reference
Remain at cohort C levels†	1,305.8 (1,279.4 to 1,325.3)	−62.0 (−78.4 to −49.2)	−4.5 (−5.7 to −3.6)
Percent reduction			
10%	1,388.4 (1,313.6 to 1,354.3)	20.5 (15.8 to 25.8)	1.5 (1.2 to 1.9)
20%	1,408.8 (1,341.4 to 1,381.4)	41.0 (31.9 to 51.6)	3.0 (2.3 to 3.8)
30%	1,429.3 (1,368.1 to 1,410.2)	61.5 (47.8 to 77.5)	4.5 (3.5 to 5.6)
Hypothetical thresholds‡			
15 $\mu g/m^3$	1,431.0 (1,406.1 to 1,458.6)	63.1 (48.7 to 80.1)	4.6 (3.6 to 5.8)
12 $\mu g/m^3$	1,455.3 (1,426.2 to 1,489.3)	87.4 (67.7 to 110.7)	6.4 (5.0 to 8.1)

For definition of abbreviations, *see* Table 2.
*Predicted outcome based on observed exposure.
†Predicted outcome if all participants in cohorts D and E had experienced exposures equal to that of participants in cohort C.
‡Predicted outcome if all participants in communities that exceeded each threshold had been assigned exposures equal to that threshold.

We observed statistically significant increases in lung growth in adolescence under hypothetical scenarios in which regional NO_2 and $PM_{2.5}$ levels were reduced by a selected percentage among all participants, as well as in dynamic interventions in which only individuals in communities above certain thresholds were intervened on and assigned pollutant concentrations equal to the threshold. This latter scenario mimicked a situation in which communities would be required to reduce levels of an air pollutant to meet an air quality standard. We additionally estimated FEV_1 would have been between 2% and 3% lower and FVC would have been between 4% and 5% lower had levels of NO_2 and $PM_{2.5}$ remained elevated as they were in 1994 to 1997 and had not experienced the observed decreases over two decades across the three cohorts.

Sensitivity analyses were previously conducted in the study from which the present mixed-effects linear regression model is derived (15). Effect estimates remained statistically significant and of similar magnitude when the sample was restricted to children with complete 4-year follow-up data or when adjusted for additional potential confounders such as tobacco exposure (*in utero*, passive, or personal), baseline asthma status, or factors related to social economic status (15). Additionally, differences in pollution effects on lung-function growth were not statistically different between children with and without asthma (15).

Although this study focused on hypothetical benefits of better lung growth from 11 to 15 years of age with reductions in regional air pollution, these benefits are likely to extend to individuals of other ages considering epidemiological studies have reported negative impacts of air pollution on younger as well as older individuals (10, 12, 25, 26). This is biologically plausible because it has been suggested that alveolarization of the lungs does not stop during childhood but continues into adulthood and thus can still be influenced by environmental factors (27). In an earlier study of older adolescents from the CHS, those who moved to cleaner communities had better lung-function growth compared with those who moved to dirtier communities (28). Additionally, researchers found reductions in ambient particulate matter were associated with slower rates of lung-function decline in a large cohort of Swiss adults (29). Therefore, improvements in air quality may also be beneficial to individuals whose lung health may have already been compromised by previous air pollution exposure.

Quantifying the benefit of improved lung function from a clinical and economic standpoint is challenging because low lung function in children is not intrinsically a treatable disease, but low lung function in children may be associated with other health outcomes such as COPD during adulthood (7). In the United States, 6.3% of adults (an estimated 15 million) have been diagnosed with COPD (30), and millions more may have the disease but be undiagnosed (31). It is currently the third leading cause of death among Americans and annual medical costs are estimated to be near 50 billion dollars by 2020 (32, 33). In one study, among participants with low FEV_1 (<80% of predicted value) before age 40 years, 26% were diagnosed with COPD later in adulthood compared with 7% of participants who had high FEV_1 (≥80% of predicted value) during early adulthood (6). Increasing lung-function growth in adolescents by reducing air pollution levels would ostensibly shift the distribution of young adults with low lung function, thus possibly reducing the number of individuals who develop COPD later in life. Additional public health and cost benefits could be assumed if low lung function is causally related to other health outcomes, including cardiovascular disease (3, 34, 35).

Assumptions are required for the findings presented here to have a causal interpretation. These assumptions are not restricted to causal inference methods and most are common to many, if not all, empirical analytic approaches but often are not specifically discussed. By purposely examining them, we provide critical information as to the level of interpretation for these findings. Conditional exchangeability presupposes adequate control for confounding and selection bias (36). The analysis focused on the effect of changing regional air pollutants in the same five communities across three different time periods, thus reducing concerns about spatial confounding. However, temporal confounding is of concern especially with the dramatic increase in Hispanic participants, but this was accounted for by inclusion of an adjustment variable for Hispanic ethnicity. Other possible temporal confounders, such as shifts in the number of asthmatic participants and socioeconomic status as measured by parental education, were previously explored but were not found to confound the association between air pollutants and lung function in this cross-cohort design (15). Participants were recruited from entire classrooms, thus minimizing the amount of selection bias in the study. Other assumptions necessary for the present analysis to have a causal interpretation include counterfactual consistency (exposure levels are a result of a well-defined intervention), positivity (all possible exposure values were observed for every confounder subgroup), correct model specification, and no interference (observations are independent; i.e., exposure to air pollutants in another community do not interfere with the effect of air pollutants in the present community) (16, 20). For correct model specification, we assumed the exposure period used in the model (i.e., 4-yr cohort period) adequately captured the biologically relevant window of susceptibly to air pollutant exposure that would impact lung function from ages 11 to 15 years. An analysis of CHS participants who moved to new communities found changes in lung function to be associated with changes in air pollution exposure within a short period of time after moving (28), supporting a short latency period between exposure and response.

A strength of this study is the use of an advanced causal inference method to estimate shifts in lung-function distribution in children under several hypothetical air pollution interventions. The interventions selected were driven by policy and results (i.e., mean lung function in the study population) are likely to be better comprehended by policymakers and the public compared with traditional regression estimates because it moved beyond simply reporting point estimates and instead estimated an answer to the question "What would have happened to lung-function growth in children from ages 11 to 15 years had their exposure to air pollution been X?" (where X is one of the various air pollutant exposure scenarios examined here). Application of causal inference methods, although growing, remains limited in health studies of air pollution (37–43). Another strength is consistent protocols across the three cohorts for measuring outcome (i.e., lung function via spirometry) and exposure (i.e., NO_2 and $PM_{2.5}$ via the same central site monitors), as well as assessing covariate information.

There are a couple of limitations to be considered in the interpretations of findings. First, high correlation between regional NO_2 and $PM_{2.5}$ in our data precluded us from including both pollutants in the same model. Thus, we are unable to disentangle the independent effects of these two pollutants or assess their interactive effect on lung function. Motor vehicles are one of the main sources of NO_2 and $PM_{2.5}$ production in Southern California. Any interventions targeting one of these exposures (e.g., reducing traffic emissions) would likely result in the reduction of the other. This makes it difficult to disentangle health effects of these two pollutants and to determine their independent impacts on health under scenarios of air pollution reduction. Single-pollutant interventions, however, most closely align with air quality standards that are set for one pollutant at a time (e.g., EPA National Ambient Air Quality Standards). Second, the relatively small number of communities in our data limits our ability to determine the exact relationship between exposure and outcome. Although our model assumed a linear relationship between each pollutant and lung function, we acknowledge there is uncertainty regarding this linearity assumption.

Conclusions

This study expands on previous work by estimating improved lung-function growth under different scenarios of reduced exposure to air pollutants. Although policies of how reductions in air pollution could be achieved were not discussed, we presented two strategies: one in which air pollutant levels were decreased by a certain percentage across all communities and another that was targeted at communities that exceeded accepted thresholds. Given the number of health outcomes associated with impaired lung function, such as COPD and cardiovascular disease, improvements in lung function in children could have long-term positive impacts on public health. ■

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