

Early-life liquefied petroleum gas cooking intervention and lung function in Guatemalan children: a randomized clinical trial

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Abstract

Rationale: Household air pollution is a risk factor for obstructive lung diseases.

Objectives: We estimated the effect of an early-life liquefied petroleum gas cooking intervention on childhood lung function.

Methods: The multi-country Household Air Pollution Intervention Network trial randomized 800 pregnant women (9 to <20 weeks gestation, 18 to <35 years) in Guatemala to receive a gas cookstove and free fuel intervention or continue cooking with biomass until the child is aged 1 year. The present analysis includes only Guatemalan children. At age 3 years, we measured lung function using oscillometry at 7–41 Hz (Tremoflo C-100, Thorasys). Outcomes were resistance and reactance at 7 Hz, area of reactance, resistance at 19 Hz, resistance difference between 7 and 19 Hz, and, as exploration, resistance across frequencies. Upper airway artifacts were removed using Tremoflo software. The effect of the intervention was estimated using linear regression models, unadjusted and adjusted (height, weight, age, sex). Children with respiratory infections during a 7-day recall period were excluded.

Results: Valid oscillometry was obtained from 525/750 (70%) 3-year-olds. Among the 225 missed oscillometry tests, 129/225 (57%) were due to the children not being willing to perform the measurements. We did not find evidence of an effect on pre-specified oscillometry outcomes. Exploratory analysis suggested that resistance across the frequency spectrum was lower in intervention than control participants (adjusted difference: -0.31 cm H₂O*s/L, 95% CI: -0.59 , -0.03).

Conclusions: Gas cooking compared to biomass cooking from mid-gestation through infancy was not associated with improved prespecified oscillometry outcomes in 3-year-old children. However, lowered resistance across all frequencies in the

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intervention arm suggests the intervention may have positively impacted airway caliber. Further studies are warranted, including exposure-response analysis and lung function trajectories.

Trial registration: Clinical trial registered at ClinicalTrials.gov (NCT 02944682).

Keywords indoor air pollution (D016902), obstructive lung diseases (D008173), embryonic and fetal development (D005314), child health (D000067576), randomized control trial (D016449)

Introduction

Chronic lung diseases cause a high burden of death and disability in low- and middle-income countries (LMICs). In 2021, chronic obstructive pulmonary disease (COPD) was responsible for 6%-9% of deaths among adults older than 55 years in LMICs, while asthma was responsible for 3%-4% of years lived with disability among children 5–14 years.¹ Household air pollution is considered a risk factor for chronic lung diseases, as indicated by meta-analyses on the effects of solid fuel use and fine particulate matter.^{2–4} As of 2020, approximately 2.8 billion people, mainly residing in LMICs, cooked daily with biomass fuel stoves.⁵

Reductions in household air pollution from cleaner cooking interventions during pregnancy and early childhood may prevent the incidence of obstructive lung disease. Per the developmental origins of health and disease (DOHaD) theory, harmful environmental exposures during periods of rapid lung development and growth may decrease lung function and have detrimental lifelong health consequences.^{6–9} Air pollution exposure may directly affect lung development or indirectly through pathways such as increased risk of low birth weight, preterm birth,^{10,11} or respiratory tract infections.^{12,13} Associated physiologic mechanisms may include changes in lung development, alteration of lung injury repair processes, oxidative stress, or epigenetic adaptations.⁹ The DOHaD theory is supported by research on perinatal exposure to tobacco smoke and ambient air pollution.^{7,14–19}

Few studies have assessed whether perinatal cookstove interventions reduce the impact of household air pollution exposure on child respiratory health.^{12,20–25} Specifically, despite substantially reducing personal exposure to fine particulate (PM_{2.5}) and carbon monoxide (CO), cookstove intervention trials have shown mixed evidence about a protective effect on pneumonia incidence.^{12,22,23,25} However, examination of lung function may reveal more subtle changes caused by air pollution, as impaired lung function may precede the clinical presentation of lung disease. Intervention studies like the Chronic Respiratory Effects of Early Childhood Exposure to Respirable PM in Guatemala²¹ and the Ghana Randomized Air Pollution and Health Study (GRAPHS)²⁰ found effects on lung function outcomes. Although only peak expiratory flow growth reached statistical significance, the Guatemalan study provided important evidence that a randomized chimney biomass stove earlier in life (before age 6 months), as opposed to later (after age 18 months), may improve lung function growth as measured by spirometry from 5 to 8 years of age.²¹ The GRAPHS study examined an improved combustion chamber biomass stove and a cleaner liquefied petroleum gas (LPG) stove intervention compared to traditional biomass cooking during pregnancy and infancy.²⁰ Oscillometry measurements from Ghana provided key insights by suggesting lower small airway resistance and higher lung compliance in children living in households cooking with LPG compared to traditional biomass, although these effects were consistent for only some of the oscillometry endpoints.²⁶ Additional evidence of the benefits of clean fuel use across

different lung function endpoints and confirmed in other populations would support household air pollution interventions early in life to prevent chronic lung disease.

The Household Air Pollution Intervention Network (HAPIN) was a multicounty randomized controlled trial conducted between 2018 and 2021 to assess the health effects of an LPG cooking intervention compared to cooking with biomass fuels from mid-pregnancy through infancy.²⁷ The intervention substantially reduced personal exposure to PM_{2.5}, CO, and black carbon,^{28,29} but we did not find effects on birth weight, severe pneumonia, or child growth up to age 1 year.^{22,30,31} We aim to estimate the effect of this early-life LPG intervention deployed at a critical stage of development on lung function at age 3 years for HAPIN participants in Guatemala, where lung function was measured. Some of the results of this study have been previously reported in the form of an abstract.³²

Methods

Study design and participants

The HAPIN trial, described previously,^{22,27,30,31} performed a stratified randomization of pregnant women in Guatemala, India, Peru, and Rwanda. The characteristics of study participants have been described before.²⁹ This analysis is restricted to Guatemala, where children had a lung function measurement at age 3 years. Briefly, in Guatemala, 800 pregnant women at <20 weeks of gestation, aged 18 to <35 years, and who exclusively cooked with biomass were randomized 1:1 to the intervention or to continue cooking as usual. The intervention consisted of a three-burner LPG cookstove available locally, free continuous supply and delivery of fuel, regular evaluation of stove condition with repairs conducted as needed, and behavior change messaging to promote safe and exclusive use of LPG until age 1 year. For the lung function study, we recruited HAPIN children in Guatemala at age 3 years from February 2022 to July 2023. Ethical review approvals and trial registration are listed in the [Supplementary Material](#).

Lung function assessment

Lung function was assessed by respiratory oscillometry to measure airflow resistance and reactance, indicators of airway narrowing and lung compliance, respectively. We used the Tremoflo C-100 Airwave Oscillometry System (THORASY Thoracic Medical Systems, Montreal, Quebec, Canada) at a pressure oscillation frequency of 7–41 Hz. Our primary outcomes were resistance at 7 Hz (R7), reactance at 7 Hz (X7), and the area above the reactance curve (AX). We selected R7 and X7 because they reflect mechanical properties of the respiratory system, with contributions from the airways, lung parenchyma, vasculature, muscles, and chest wall, and AX because it may be a more sensitive measure of compliance than reactance at any single frequency.^{33–35} Secondary endpoints were resistance at 19 Hz (R19), as elevations may

indicate central airway narrowing, and the frequency dependence of resistance (R7–R19), which may provide information on smaller airways. Resistance measures pooled across prime frequencies (R) were included as an exploratory outcome. We speculated that R could be sensitive to changes in more locations of the respiratory system than single frequencies.

A team of two field workers, including a nurse, visited participants in their homes. They collected two consecutive oscillometry tests, and for each test, they collected four to eight 20-s oscillometry measurements. Measurement artifacts, defined as ± 3 standard deviations from the average of R7, or implausible negative R values, were automatically removed by the Thorasy software. Measurements with $\geq 70\%$ of data points remaining after artifact filtering were considered valid. The first three valid measurements with a coefficient of variation $< 15\%$ for R7 were selected and averaged to obtain test summaries, but if this criterion was not met, we averaged all valid measurements. To reduce random error, the average of the two consecutive tests conducted during the same home visit was used in the analysis. Details about respiratory oscillometry are provided in the [Supplementary Material](#). During the visit, children were screened for acute lower respiratory infections (ALRI) in the prior 7 days. Children with ALRI were excluded from the analyses as their lung function may not represent their healthy baseline, which was this study's goal. The screening is explained in the [Supplementary Material](#). A subset of children who were unable to perform a valid oscillometry measurement at age 3 years was revisited at age 4. Findings at age 4 years are reported in sensitivity analyses only.

Statistical analysis

The intention-to-treat effect of the intervention was estimated from the unadjusted difference in oscillometry outcomes between study arms. We used linear regression models for primary and secondary outcomes. We modeled R pooled across frequencies using linear mixed-effects regression models with a quadratic b-spline for frequency with knots at 13, 21, and 31 Hz, and random child intercept and frequency slope. This model was selected using the Akaike information criterion ([Table S1](#) in the [online supplementary material](#)). Power calculations for unadjusted comparisons of primary outcomes between study arms are shown in the [Supplementary Material](#).

In addition, we aimed to estimate the effect on lung function independently of lung size by adjusting for height and weight, and, to improve model precision, we also adjusted for age and sex (adjusted models). To reduce bias potentially introduced by conditioning on intermediate variables and missing outcome data, we used marginal structural models (fully adjusted models) to further adjust for recent acute illness, gestational age at randomization, pre-randomization sociodemographic variables, and secondhand smoke using methods presented by Hernan and Robins.³⁶ Variable selection was based on causal assumptions represented in the directed acyclic graph in [Figure S1](#) (see [online supplementary material](#) for a color version of this figure). Missing covariate data were imputed using multivariate imputations by chained equations. The statistical details of the fully adjusted models are described in the [Supplementary Material](#).

Sensitivity analysis

Sensitivity analyses were performed to explore the estimated intervention effect using an alternative oscillometry quality control method ([Table S2](#)), including children with ALRI within 7 days of oscillometry, and including oscillometry measurements collected at age 4 years (age 4 measurements were collected from children with missing measurements at age 3 years).

Subgroup analysis

We also examined effect modification by sex, and the timing of the intervention (before versus after median gestational age at randomization) using marginal structural models with interaction terms.

All analyses were implemented in R statistical software (version 4.4.2).

Results

Participant follow-up

[Figure 1](#) shows the population enrolled, randomized, and followed up during the study. Out of 800 randomized pregnant women, 262 (66%) children in the intervention group and 263 (66%) in the control group were included in the final analytical sample. Of the 750 three-year-old children, 225 (30%) were censored; the most common reasons were unwillingness to perform oscillometry ($n=129$), migration ($n=37$), and lack of caregiver consent ($n=26$). Of the participants in the analytical sample, 517 (98%) had valid results on both oscillometry tests collected consecutively during the household visit, while 8 (2%) had valid results for only one test. Of the 1042 tests, 1028 (99%) had three measures with an R7 coefficient of variation $\leq 15\%$.

Pregnant women included in the analytical sample had similar characteristics to those not included ([Table S3](#)). However, children included compared to those not included were more likely to be male (55% vs. 44%), less likely to be born preterm (4.8% vs. 10%), heavier at birth (median: 2910 g vs. 2736 g), and taller (median: 88.3 cm vs. 87.0 cm) and heavier (median: 12.7 kg vs. 12.3 kg) at age 3 years.

Participant characteristics

The intervention and control participants were similar at baseline ([Table 1](#)). Summary statistics of the covariates used in regression analysis after imputation are shown in [Table S4](#).

Mean exposure to particulate matter < 2.5 micrometers in diameter ($PM_{2.5}$) was 141.9 (SD: 129.3) $\mu g m^{-3}$ at baseline. Post intervention, mean exposure to $PM_{2.5}$ in the intervention arm was lower by 78% (132.2 vs. 29.6 $\mu g m^{-3}$) during pregnancy and 82% (157.6 vs. 28.3 $\mu g m^{-3}$) during infancy compared to the control arm. After the intervention period, many households in the intervention group returned to using biomass cooking fuel, and some in the control group used LPG stoves. At age 2 years, 13% of the intervention group and 5% of the control group reported the LPG stove as their

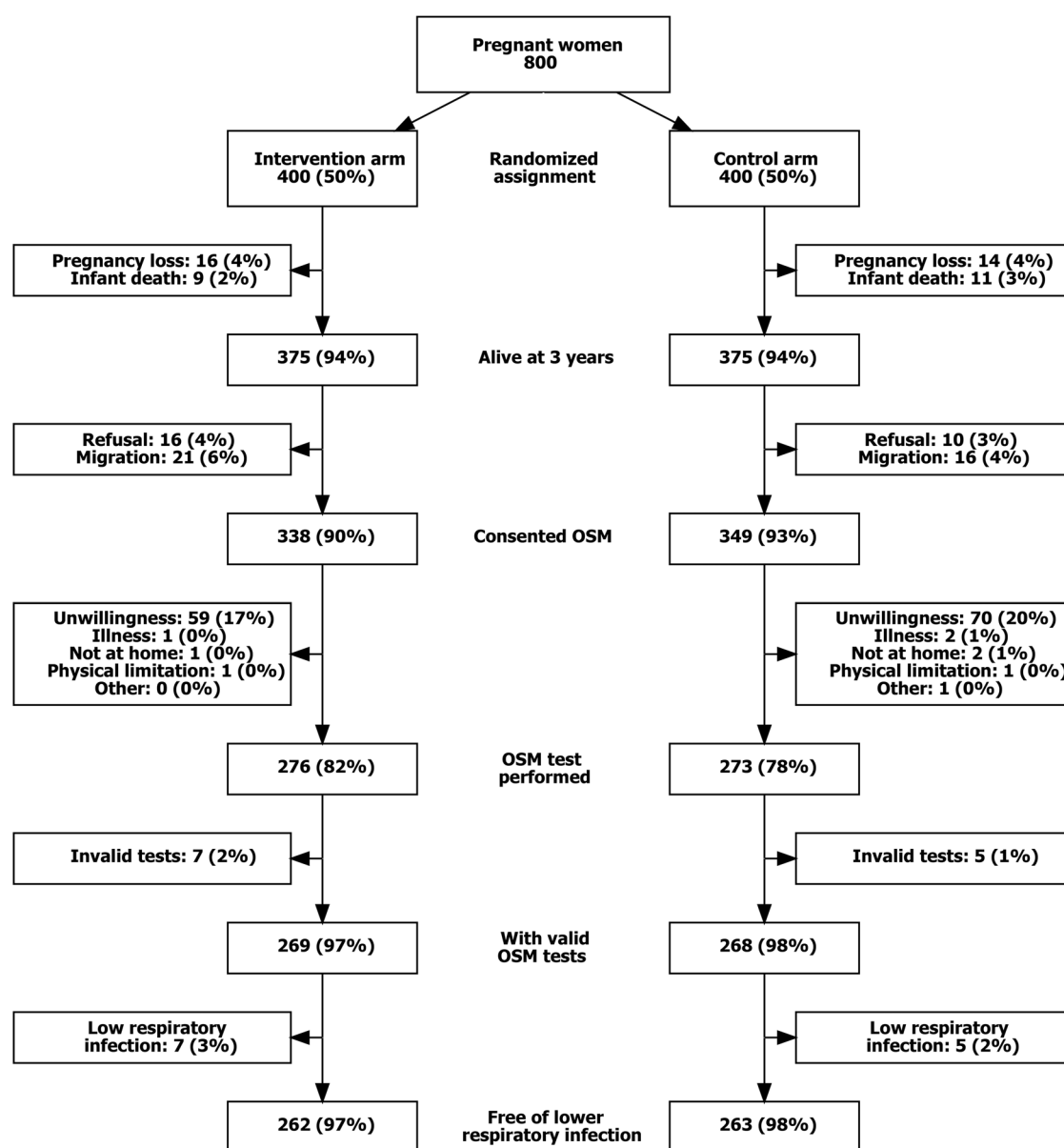


Figure 1 Trial profile. OSM: respiratory oscillometry.

primary stove. Similarly, at age 3 years, 21% of the intervention group and 9% of the control group reported the LPG stove as their primary stove (Table 1).

Oscillometry results

The mean (SD) of R7, X7, AX, R19, R7–R19, and R in $\text{cmH}_2\text{O}^*\text{s/L}$ were 13.51 (2.57), –5.56 (1.54), 92.73 (39.04), 11.45 (2.26), 2.06 (1.15), and 11.38 (2.49), respectively. We did not find evidence that the LPG intervention affected R7, X7, AX, R19, or R7–R19, but R7 and R19 were consistently lower across adjustment sets in the intervention group compared to the control (Table 2). In contrast, R was statistically significantly lower in the intervention than in the control children (Figure 2). There was no evidence of interaction between oscillometry frequency and the trial arm. The results were not sensitive to the oscillometry quality control method (Figures S2 and S3, see online supplementary material for a color

version of these figures), the inclusion of children with ALRI in the past 7 days (Table S5), or the inclusion of oscillometry tests collected at age 4 years (Table S6 and Figure S3, see online supplementary material for a color version of this figure). No effect modification was observed by earlier (≤ 15.6 weeks) versus later (> 15.6 weeks) gestational age at the time of randomization or sex when evaluating the primary or secondary outcomes (Table 3).

Discussion

In this trial, we did not observe statistically significant differences in the primary or secondary lung function outcomes in 3-year-old children living in households randomized to receive an LPG cookstove intervention versus those who continued using biomass fuel from mid-gestation to age 1 year, but the effect estimates for R7 and R19 were in the direction expected with better lung function in the intervention group. Furthermore, the intervention

Table 1 Characteristics of study participants in the *analytical dataset*.

Characteristic	Intervention N = 262	Control N = 263	P value ^a
Baseline			
Valid maternal PM _{2.5} exposure, mean (SD)	233 (89)	251 (95)	.009
Maternal PM _{2.5} exposure in $\mu\text{g m}^{-3}$ (n = 484), ^b mean (SD)	146 (113)	138 (143)	.487
Maternal age in years, n (%)			.737
[18–20]	41 (16)	37 (14)	
[20–25]	105 (40)	104 (40)	
[25–30]	80 (31)	77 (29)	
[30–35]	36 (14)	45 (17)	
Maternal education, n (%)			.847
None or some primary	120 (46)	126 (48)	
Primary or some secondary	107 (41)	101 (38)	
Secondary or more	35 (13)	36 (14)	
Socioeconomic tertiles, ^c n (%)			.654
Low	90 (34)	81 (31)	
Mid	86 (33)	94 (36)	
High	89 (33)	88 (33)	
Second-hand smoke in the household, n (%)	14 (5)	18 (7)	.592
Household food insecurity, ^d n (%)			.985
Food secure	139 (53)	139 (53)	
Mild food insecurity	85 (32)	88 (33)	
Moderate or severe food insecurity	34 (13)	33 (13)	
Missing	4 (2)	3 (1)	
Number of siblings in the house, n (%)			.242
0	84 (32)	65 (25)	
1	74 (28)	74 (28)	
2	51 (19)	60 (23)	
3 or more	53 (20)	64 (24)	
Male, n (%)	148 (56)	138 (52)	.403
Intervention			
Gestation at baseline in weeks, med [IQR]	14 [12, 17]	14 [12, 16]	.223
Gestation at randomization in weeks, med [IQR]	16 [14; 18]	15 [13, 18]	.124
Gestation at stove installation in weeks, med [IQR]	17 [15, 21]	-	-
Pregnancy trial period			
Valid maternal PM _{2.5} exposure ^e	259 (99)	254 (97)	.146
Maternal PM _{2.5} exposure in $\mu\text{g/m}^3$ (n = 513), ^e mean (SD)	30 (19)	132 (99)	<.001
Preterm birth, ^f n (%)	13 (5)	12 (5)	.992
Gestation at birth in weeks, med [IQR]	39 [38, 40]	39 [38, 40]	.690
Low birth weight, ^g n (%)			.733
Yes	32 (12)	35 (13)	
No	226 (86)	222 (84)	
Missing	4 (2)	6 (2)	
Birth weight in grams (n = 515), ^h med [IQR]	2900 [2678, 3168]	2913 [2698, 3168]	.946
Infancy trial period			
Valid infant PM _{2.5} exposure	224 (85)	228 (87)	.787
Infant PM _{2.5} exposure in $\mu\text{g m}^{-3}$ (n = 452), ⁱ mean (SD)	28 (21)	158 (157)	<.001
Diagnosis of severe pneumonia, ^j n (%)	23 (9)	15 (6)	.234
Up-to-date pentavalent vaccination at trial exit, ^k n (%)			.586
Yes	244 (93)	247 (94)	
No	15 (6)	11 (4)	
Missing	3 (1)	5 (2)	
Up-to-date pneumococcal conjugate vaccination at trial exit, ^l n (%)			.747
Yes	245 (94)	250 (95)	
No	8 (3)	6 (2)	
Missing	9 (3)	7 (3)	
Child characteristics at age 2 years			
Primary stove, n (%)			.002

(Continued)

Table 1 Continued.

Characteristic	Intervention N = 262	Control N = 263	P value ^a
Open fire	159 (61)	160 (61)	
Chimney stove	49 (19)	69 (26)	
LPG stove	35 (13)	12 (5)	
Electric stove	1 (0)	0 (0)	
Missing	18 (7)	22 (8)	
Child characteristics and health at oscillometry (age 3 years)			
Primary stove, n (%)			<.001
Open fire	166 (63)	188 (71)	
Chimney stove	40 (15)	52 (20)	
LPG stove	56 (21)	23 (9)	
Age in years, med [IQR]	3.1 [3.1, 3.2]	3.1 [3.1, 3.2]	.549
Height in cm, mean (SD)	88 (4)	88 (4)	.536
Weight in kg, mean (SD)	13 (1)	13 (1)	.550
Cough in the last 7 days,^mn (%)	46 (18)	50 (19)	.750
Common cold in the last 7 days,ⁿn (%)			.908
Yes	46 (16)	46 (17)	
No	218 (83)	216 (82)	
Missing	1 (0)	1 (0)	
Fever in the last 7 days,ⁿn (%)			.532
Yes	10 (4)	14 (5)	
No	251 (96)	249 (95)	
Missing	1 (0)	0 (0)	
Diarrhea in the last 7 days,ⁿn (%)			.028
Yes	31 (12)	17 (6)	
No	231 (88)	244 (93)	
Missing	0 (0)	2 (1)	

^aFor categorical or binary variables, the *P* values were calculated using a chi-squared or Fisher test; for continuous variables, the *P* values were calculated using *t*-tests or the Mann-Whitney *U* test.

^bPreintervention 24-h personal exposure to particulate matter with aerodynamic size <2.5 μm.

^cSocioeconomic status was calculated as the first component in a principal component analysis of water source, sanitation, electricity, housing materials, and ownership of 24 household assets.

^dHousehold food insecurity was measured using the Food Insecurity Experience Scale (FIES) applied with a 30-day reference period.

^eAverage of two 24-h personal exposure measurements of PM_{2.5} collected at 24–28 and 32–36 weeks of gestation.

^fBirth <37 weeks gestation.

^gBirthweight <2500 g.

^hWeight measured by study personnel within 24 h of birth. If missing, the weight was obtained from health services records.

ⁱAverage of three 24-hour indirect exposure measurements of PM_{2.5} collected at 3, 6, and 12 months old.

^jCase definition adapted from the World Health Organization's Integrated Management of Childhood Illness to improve specificity and objectivity.

^kThree doses by age 1 year.

^lTwo doses by age 1 year.

^mCaregiver report, but when the caregiver reported the absence of the cough, the cough was assessed by observation from the study nurse.

ⁿCaregiver report.

reduced lower airway resistance as measured by pooling resistance across the prime frequencies between 7 and 41 Hz (*R*, our exploratory outcome). This finding suggests a larger airway caliber in the intervention than in the control children. Strengths of our study included randomization, characterization of personal exposure to air pollutants, and use of a lung function technique feasible in young children. The cleaner cookstove and behavioral intervention had high fidelity and adherence, with reports of running out of LPG in only 50 of 19 623 (0.3%) household visits, and traditional stove use among intervention households occurring only on a median of 0.4% of the monitored household-days.³⁷ Given the high adherence, we did not pursue per-protocol analysis.

Our study is novel in modeling the airway resistance across pressure oscillation frequencies. A study in Austria in school-aged children also used resistance from multiple frequencies. However, they measured oscillometry from 4 to 30 Hz and used the data

points to estimate the hypothetical resistance at 0 Hz (*R*₀).³⁸ They found *R*₀ to be positively associated with concentrations of ambient particulate matter less than 10 (PM₁₀), 2.5 (PM_{2.5}), and 1 (PM₁) μm in diameter near the school in the 8 h prior to lung function assessment.³⁹ We hypothesize that outcomes based on resistance across a range of frequencies may be more sensitive to the effects of air pollution than resistance at single frequencies.

Our results can be compared with those of the GRAPHS trial, in which 4-year-old children whose mothers received the LPG intervention at an average gestational age of 16 weeks through age 1 year had lower *R*₅–*R*₂₀ and higher (less negative) *X*₅ compared to controls whose mothers continued cooking with biomass fuels.²⁰ These findings were interpreted as reflecting lower peripheral airway resistance. In our study, we did not find evidence of an effect of the LPG intervention on similar endpoints (*R*₇–*R*₁₉, *X*₇) measured at age 3 years; however, we did find evidence of lower airway resistance in the intervention children compared to the

Table 2 Effect of LPG intervention on lung oscillometry at age 3 years in cmH₂O*s/L.

Outcome	Intervention Mean (SD)	Control Mean (SD)	Unadjusted difference	Adjusted difference ^a	Fully adjusted difference ^b
			(95% CI) N = 525	(95% CI) N = 525	(95% CI) N = 525
R7	13.43 (2.44)	13.58 (2.70)	-0.15 (-0.59, 0.29)	-0.20 (-0.61, 0.22)	-0.27 (-0.76, 0.23)
X7	-5.54 (1.44)	-5.58 (1.63)	0.04 (-0.22, 0.31)	0.08 (-0.17, 0.33)	0.03 (-0.29, 0.35)
AX	92.57 (35.95)	92.88 (41.96)	-0.31 (-7.01, 6.39)	-1.30 (-7.63, 5.04)	0.92 (-8.10, 9.93)
R19	11.32 (2.12)	11.58 (2.38)	-0.26 (-0.65, 0.12)	-0.29 (-0.65, 0.07)	-0.35 (-0.77, 0.07)
R7-R19	2.11 (1.10)	2.00 (1.19)	0.11 (-0.08, 0.31)	0.10 (-0.09, 0.30)	0.08 (-0.13, 0.29)
R	11.25 (2.40)	11.50 (2.58)	-0.29 (-0.59, 0.00)	-0.31 (-0.60, -0.03)	-0.31 (-0.54, -0.06)

^aEffect independent of lung size by adjustment for the child's age, sex, height, and weight.

^bEffect independent of lung size and addressing potential bias by using inverse probability weighting to adjust for child's age, sex, height, weight; presence of cough, common cold, fever, or diarrhea in the last week; gestational age at randomization; and pre-randomization socioeconomic score, food insecurity score, exposure to secondhand smoke, maternal education, maternal age, and number of siblings.

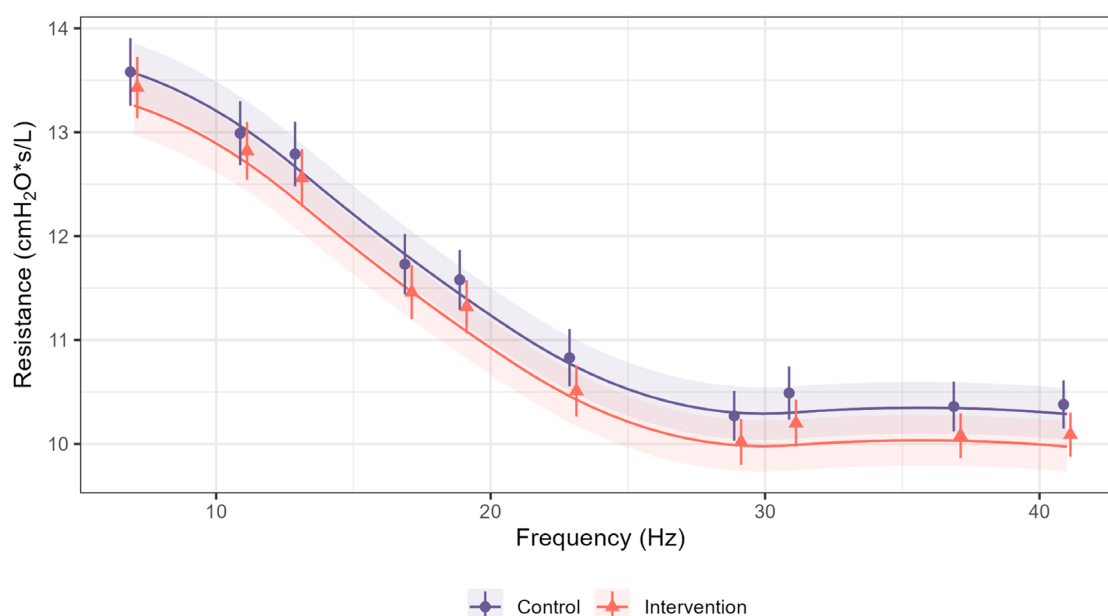


Figure 2 Resistance measures by trial arm and frequency. The intervention arm is represented in purple circles and the control arm in orange triangles. The points represent observed child averages and 95% confidence intervals. The lines show predicted resistance from a model with quadratic B-splines for frequency with knots at 13, 21, and 31 Hz, a random subject intercept, a random subject slope for frequency, and adjusted for the child's sex, age, height, and weight. Predictions with variables held at their means or modes: males, age 3.1 years, height 88.2 cm, and weight 12.9 kg.

controls when evaluating *R*, our exploratory endpoint. The qualitatively different findings in Ghana and Guatemala could be due to different mixtures of air pollutants, different distributions of effect modifiers in the studied populations, different mechanisms by which air pollution could have affected lung function (such as differences in pneumonia incidence), differences in oscillometry measurement procedures and quality control, or chance.

There is reason for caution against overinterpreting oscillometry endpoints in relation to physiological changes. Notable contrasts exist between GRAPHS and our study in the direction of the resistance effect estimates. GRAPHS reported higher *R*₂₀ and no change in *R*₅ among children in the intervention arm, while in our study, both *R*₇ and *R*₁₉ were lower, with the decrease in *R*₇ being smaller than in *R*₁₉; these changes, however, were not statistically significant. Both patterns are inconsistent with the common interpretation of *R*₅–*R*₂₀ and *R*₇–*R*₁₉ as a surrogate for small airway resistance. This interpretation assumes that resistance at low

frequencies represents the total respiratory system and is therefore elevated in the presence of small airway narrowing, while resistance at middle frequencies represents the central airways. In neither study was resistance at the low frequency more affected than resistance at the middle frequency. *R*₅–*R*₂₀ and *R*₇–*R*₁₉ can also result from upper airway shunting or heterogeneous airway narrowing, and can be impacted by glottic artifacts or poor technique.

Spirometry is the most standard lung function technique for evaluating obstructive lung disease; however, we chose oscillometry because it is effort independent and therefore feasible for young children.^{40,41} In addition, oscillometry may be equally or more sensitive for detecting small airway obstruction,^{35,42–44} and may pinpoint the location of an obstruction to the central or small airways.^{35,45} Although oscillometry and spirometry measure different aspects of respiratory mechanics, concordant effects on children's lung function have been reported. In a Swedish study,

Table 3 Effect of the LPG intervention on lung oscillometry by subgroups.

OSM endpoint	Subgroup	Intervention		Control		Effect estimate (95% CI) ^b	Interaction term (95% CI) ^b
		N	Mean (SD) ^a	N	Mean (SD) ^a		
By median gestational age at randomization							
R7	≤15.6 weeks	128	13.51 (2.53)	141	13.54 (2.63)	−0.17 (−0.85, 0.51)	0.20
	>15.6 weeks	134	13.36 (2.35)	122	13.64 (2.79)	−0.37 (−1.08, 0.34)	(−0.78, 1.17)
X7	≤15.6 weeks	128	−5.58 (1.42)	141	−5.57 (1.68)	0.01 (−0.39, 0.41)	−0.03
	>15.6 weeks	134	−5.50 (1.47)	122	−5.59 (1.58)	0.05 (−0.44, 0.53)	(−0.65, 0.58)
AX	≤15.6 weeks	128	93.92 (36.94)	141	93.31 (41.77)	0.06 (−10.24, 10.37)	−1.75
	>15.6 weeks	134	91.28 (35.07)	122	92.38 (42.34)	1.81 (−12.49, 16.12)	(−18.94, 15.45)
R19	≤15.6 weeks	128	11.43 (2.20)	141	11.61 (2.26)	−0.26 (−0.84, 0.31)	0.16
	>15.6 weeks	134	11.21 (2.04)	122	11.56 (2.53)	−0.43 (−1.04, 0.18)	(−0.67, 1.00)
R7-R19	≤15.6 weeks	128	2.08 (1.15)	141	1.93 (1.14)	0.09 (−0.20, 0.39)	0.03
	>15.6 weeks	134	2.15 (1.06)	122	2.08 (1.24)	0.06 (−0.25, 0.37)	(−0.39, 0.46)
R	≤15.6 weeks	128	11.33 (2.46)	141	11.51 (2.48)	−0.26 (−0.60, 0.07)	0.08
	>15.6 weeks	134	11.17 (2.33)	122	11.50 (2.70)	−0.35 (−0.69, −0.01)	(−0.39, 0.56)
By sex							
R7	Females	114	13.72 (2.54)	125	13.83 (2.67)	−0.37 (−1.11, 0.36)	0.21
	Males	148	13.21 (2.34)	138	13.36 (2.72)	−0.16 (−0.84, 0.52)	(−0.79, 1.22)
X7	Females	114	−5.59 (1.37)	125	−5.73 (1.76)	0.18 (−0.32, 0.68)	−0.30
	Males	148	−5.50 (1.50)	138	−5.44 (1.50)	−0.12 (−0.51, 0.27)	(−0.92, 0.32)
AX	Females	114	93.26 (35.06)	125	93.61 (42.90)	0.83 (−13.65, 15.31)	0.22
	Males	148	92.04 (36.74)	138	92.21 (41.24)	1.05 (−9.35, 11.45)	(−17.34, 17.77)
R19	Females	114	11.68 (2.21)	125	11.88 (2.23)	−0.39 (−1.01, 0.23)	0.09
	Males	148	11.04 (2.01)	138	11.32 (2.49)	−0.30 (−0.87, 0.27)	(−0.75, 0.93)
R7-R19	Females	114	2.04 (1.10)	125	1.96 (1.23)	0.01 (−0.31, 0.33)	0.12
	Males	148	2.17 (1.11)	138	2.04 (1.15)	0.14 (−0.14, 0.42)	(−0.30, 0.55)
R	Females	114	11.56 (2.49)	125	11.72 (2.48)	−0.15 (−0.53, 0.23)	−0.27
	Males	148	11.01 (2.29)	138	11.30 (2.65)	−0.41 (−0.70, −0.13)	(−0.73, 0.20)

^aIn cmH₂O*S/L.^bLinear model for oscillometry endpoint adjusted by inverse probability weighting for the child's age, sex, height, and weight; presence of cough, common cold, fever, or diarrhea in the last week; gestational age at randomization; and pre-randomization socioeconomic score, food insecurity score, number of siblings in the home, maternal education, maternal age, and exposure to second hand smoke. Confidence intervals calculated using 1000 bootstrap resampling.

adolescents whose mothers smoked during pregnancy had a lower ratio of forced expiratory volume over 1 s to forced vital capacity (FEV₁/FVC) and higher R5, R5–R20, and AX compared to adolescents of mothers who did not smoke.⁴⁶

Several possible reasons exist for the lack of effects in our primary and secondary outcomes. First, although the intervention substantially reduced personal exposures to measured pollutants during the trial period, the exposure reductions may have been insufficient to detect certain adverse effects of air pollution on lung function. Despite a mean (SD) exposure to PM_{2.5} of 132 (99) versus 30 (19) µg m^{−3} during pregnancy and 158 (157) versus 28 (21) µg m^{−3} during infancy in the control versus intervention arms,^{28,29} it is possible that a lower exposure in the intervention group would have increased power to detect an effect on primary and secondary outcomes. This explanation is particularly likely if the exposure–response relationship is steeper at lower exposures, as some studies have suggested for effects on ALRI.⁴⁷

Second, women received the intervention at a median gestational age of 17.4 weeks, corresponding to the end of the pseudoglandular stage (5–17 weeks) or the beginning of the canalicular stage (16–26 weeks) of lung development. At this time, the trachea, bronchi, terminal bronchioles, pulmonary buds, pulmonary vein, and pulmonary artery may have already formed. However, the respiratory bronchioles, alveolar ducts, and alveoli are unlikely to have formed yet. An earlier intervention may have

influenced the formation and branching of the conducting airways, resulting in permanent structural differences observable at age 3 years. GRAPHs reported sensitive prenatal exposure windows to carbon monoxide for R5 and R20 at <20 weeks of gestation, for X5 and R5–R20 at >32 weeks of gestation, and no window for AX.²⁰ In contrast, we did not detect differences in the effect on primary or secondary outcomes by timing of the intervention. However, stratified analyses may have been underpowered to detect effects.

Third, our intervention ended when the children reached 1 year, after which most intervention participants reverted to using biomass cookstoves and fuel, and a few control participants adopted LPG. Although substantial lung development has occurred by 1 year, airways continue to grow, and alveoli continue to multiply and increase in size and complexity until early adulthood, offering an opportunity for catch-up growth.⁴⁸ Air pollution exposure during the potentially susceptible period of ages 1–3 years could have dampened the benefits gained in the intervention group and improved the lung function of the controls, reducing the contrast between groups at age 3. The plasticity of the lungs after infancy was illustrated in a longitudinal lung function study in which children exposed to tobacco smoke during pregnancy had reduced lung function at 1 year of age, but by 6 years, lung function largely recovered.⁴⁹ Similarly, a birth cohort from South Africa reported impaired airway resistance at age 3 years associated with

postnatal exposure to PM₁₀, but not with prenatal exposure.⁵⁰ Evaluating the impact of more recent exposures may elucidate effects on lung function trajectories, and future research should aim to assess lung function earlier after an intervention.

Our study had limitations. First, about half of the infancy period (December 2018 to June 2021) of the children in the study coincided with the COVID-19 pandemic. During the pandemic, a reduction in the incidence of pneumonia of 77% was observed in our study population.²² A decreased risk of pneumonia and other respiratory infections may have blocked an important pathway between air pollution and lung function. Thus, in our study population, the effect may be smaller and difficult to detect or may not have affected the same oscillometry endpoints as we would have observed in the absence of the pandemic.

Furthermore, of the 800 pregnant women originally randomized, 525 (66%) were included in our analytical dataset. Losses were primarily due to children's unwillingness to perform the oscillometry test ($n=129$). Other children ($n=12$) were excluded from the analysis because they had had ALRI in the 7 days before testing, and we were unable to visit them once they recovered. Missing outcome data were balanced across trial arms; however, children in the analytical sample differed from those not included in some characteristics that may have hindered our ability to detect effects of air pollution. The analytical sample underrepresented females (a possible effect modifier), children who experienced an adverse pregnancy outcome (a mediator), and children of larger size, yielding a population that may have a lower susceptibility to the effects of air pollution.^{9,13,24} Consequently, our findings may not be generalizable to the target population.⁵¹ We corrected for possible selection bias using marginal structural models with stabilized weights for the inverse probability of not being censored (fully adjusted model). Our marginal structural models did not change the results. In a sensitivity analysis, we added 83 children who had oscillometry tests at 4 years of age and obtained similar results.

Second, while there are many advantages to using oscillometry, uncertainty is involved in the measurements. A current disadvantage of oscillometry over spirometry is that differences in resistance and reactance are not easily interpreted in terms of clinical significance because normative reference values that can be applied to our population are not yet available.^{33,52} In addition, correlations between physiological changes and specific oscillometry endpoints, remain to be fully understood.³³ Moreover, the presence of upper airway artifacts in our measurements could have reduced the sensitivity to detect the effects of air pollution. We addressed this concern by applying two alternative quality control methods, and both yielded similar results. However, we cannot rule out the possibility that subtle artifacts may have remained after any quality control and hampered our ability to measure the lungs. Lastly, we did not include bronchodilator reversibility testing or intra-breath (single frequency) oscillometry testing, which may improve the sensitivity of detecting differences in airway caliber.^{53,54}

Conclusion

In our randomized controlled trial comparing LPG cooking during pregnancy and infancy with cooking using biomass fuels, the intervention did not improve the pre-specified lung function

outcomes in 3-year-old children; however, the resistance effect estimates were consistent with better lung function in the intervention group. Furthermore, children in the intervention arm had significantly lower airway resistance pooled over prime frequencies, consistent with less airway narrowing compared to those in the control group. Future research should investigate whether gains in lung function are reversible upon returning to elevated exposure levels and whether sustained cleaner cookstove interventions, delivered earlier in pregnancy and maintained throughout childhood, are associated with improved childhood respiratory health.

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Author contributions

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Supplementary material

Supplementary material is available at *Annals of the American Thoracic Society* online.

Conflicts of interest

Please see the ICMJE disclosure forms, which have been provided as [supplementary material](#).

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Data availability

Deidentified data associated with these analyses are available through the Emory University Dataverse open data repository (<https://dataverse.unc.edu/dataverse/Emory>) and are linked to the <https://doi.org/10.15139/S3/EOBLWZ>.

Artificial intelligence disclaimer

During the preparation of this work, the author did not use generative AI or AI-assisted technologies.

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