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Association of ambient and household air pollution with lung function in young adults in an peri-urban area of South-India: A cross-sectional study

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ABSTRACT

Objective: Although there is evidence for the association between air pollution and decreased lung function in children, evidence for adolescents and young adults is scarce. For a peri-urban area in India, we evaluated the association of ambient $PM_{2.5}$ and household air pollution with lung function for young adults who had recently attained their expected maximum lung function.

Methods: We measured, using a standardized protocol, forced expiratory volume in the first second (FEV_1) and forced vital capacity (FVC) in participants aged 20–26 years from the third follow-up of the population-based APCAPCS cohort (2010–2012) in 28 Indian villages. We estimated annual average $PM_{2.5}$ outdoors at residence using land-use regression. Biomass cooking fuel (a proxy for levels of household air pollution) was self-reported. We fitted a within-between linear-mixed model with random intercepts by village, adjusting for potential confounders.

Results: We evaluated 1,044 participants with mean age of 22.8 (SD = 1) years (range 20–26 years); 327 participants (31%) were female. Only males reported use of tobacco smoking (9% of all participants, 13% of males). The mean ambient $PM_{2.5}$ exposure was 32.9 (SD = 2.8) $\mu g/m^3$; 76% reported use of biomass as cooking fuel. The adjusted association between 1 $\mu g/m^3$ increase in $PM_{2.5}$ was −27 ml (95% CI, −89 to 34) for FEV_1 and −5 ml (95% CI, −93 to 76) for FVC. The adjusted association between use of biomass was −112 ml (95% CI, −211 to −13) for FEV_1 and −142 ml (95% CI, −285 to 0) for FVC. The adjusted association was of greater magnitude for those with unvented stove (−158 ml, 95% CI, −279 to −36 for FEV_1 and −211 ml, 95% CI, −386 to −36 for FVC).

Conclusions: We observed negative associations between ambient $PM_{2.5}$ and household air pollution and lung function in young adults who had recently attained their maximum lung function.

1. Introduction

Air pollution is a major risk factor for non-communicable diseases worldwide (Murray et al., 2020). The lungs are directly exposed to the inhaled air, thus subject to the harmful effects of particulate matter (PM), toxic gases and other constituents of polluted air (Adam et al.,

Abbreviations: APCAPCS, Andhra Pradesh Children and Parent Study; BMI, body mass index; CVD, cardiovascular diseases; FEV_1, forced expiratory volume in the first second; FVC, forced vital capacity; HAP, household air pollution; LMIC, low-and-middle income countries; LUR, land-use regression; NIN, National Institute of Nutrition; PM, particulate matter; $PM_{2.5}$, particulate matter with a mean aerodynamic diameter of 2.5 μm or less; SLI, Standard of Living Index.

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2015; Götschi et al., 2008; Paulin and Hansel, 2016; Schraufnagel et al., 2019). There has been consistent evidence between high levels of air pollution and increased incidence and exacerbations of chronic respiratory diseases (Götschi et al., 2008; Guo et al., 2018; Murray et al., 2020; Paulin and Hansel, 2016). Additionally, there is evidence for high levels of air pollution and worse lung function, particularly for short-term effects and also for long-term exposure (Adam et al., 2015; Edginton et al., 2019; Götschi et al., 2008; Guo et al., 2018; Int Panis et al., 2017; Rice et al., 2015).

There are several gaps to be addressed in the association between long-term exposure to air pollution and lung function (Götschi et al., 2008; Guo et al., 2018; Paulin and Hansel, 2016). There is limited data from low-and-middle income countries and the majority of studies for long-term exposure come from areas with relatively low ambient air pollution (Adam et al., 2015; Guo et al., 2018). Additionally, there is consistent evidence for worse lung development in children exposed to high levels of air pollution, but there are few studies for adolescents; those show inconsistent results (Cai et al., 2020; Fuertes et al., 2015; Gauderman et al., 2004; Milanzi et al., 2018; Schultz et al., 2016). It is largely unknown whether the harmful effect observed during childhood could be compensated during the expansion of lung capacity during puberty and adolescence (Cai et al., 2020; Fuertes et al., 2015; Götschi et al., 2008).

While more than 3 billion individuals still rely on biomass fuel for cooking and heating, the evidence regarding household air pollution (HAP) and lung function is limited (Balmes, 2019; Raju et al., 2020; Sood et al., 2018). There is consistent evidence for increased risk of respiratory infections in children, particularly pneumonia, and some evidence for tuberculosis and chronic respiratory diseases in adults (Balmes, 2019; Jindal et al., 2020; Raju et al., 2020). However, few studies evaluated the association between HAP and lung function in children and adolescents (Aithal et al., 2021; Sood et al., 2018), and adults (Amaral et al., 2018; Siddharthan et al., 2018; Sood et al., 2018). Overall, these studies are limited by a small sample size (Patel et al., 2018), occupational exposure or specific populations (Dutta et al., 2021; Singh et al., 2017), and lack of adjustment for ambient air pollution (Simkovich et al., 2019).

Lung function growth and the maximum attained lung function, achieved at around 18 years in females and 20 years in males (Kohansal et al., 2009; Rennard and Drummond, 2015), are among the main determinants of lung function levels and the occurrence of chronic respiratory diseases in adulthood (Agustí et al., 2017; Rennard and Drummond, 2015). For a peri-urban area in India, we evaluated the association between ambient $PM_{2.5}$ and HAP with lung function for young adults who had recently attained their expected maximum lung function. We hypothesized that both ambient $PM_{2.5}$ and HAP are associated with worse lung function.

2. Methods

2.1. Study design

We conducted a cross-sectional, population-based analysis in the third follow-up of the Andhra Pradesh Children and Parent Study (APCAPS) cohort (2010–2012) (Kinra et al., 2014). APCAPS is a large prospective, intergenerational cohort that began with the long-term follow-up of the Hyderabad Nutrition Trial (1987–1990). We used data from the third follow-up, which surveyed 6,944 participants situated in 28 villages in a peri-urban area south of Hyderabad, India (eFigure 1). We selected the index children (n = 1,360 participants), traced from the Hyderabad Nutrition Trial, who were young adults (20–26 years) at the third follow-up, in order to estimate the effect of air pollution right after the peak of lung function development.

APCAPS was approved by the London School of Hygiene & Tropical Medicine (London, UK) and the National Institute of Nutrition (NIN) (Hyderabad, India). Signed consent forms were obtained from all participants.

2.2. Data collection

Data were collected at clinics in the 28 APCAPS villages. Following standard operating procedures, data were collected via standardized questionnaires by trained interviewers, assessing demographic, socioeconomic status [education, occupation, Standard of Living Index (SLI)] (Kinra et al., 2014), health behaviours (smoking, environmental tobacco smoke, and physical activity) and anthropometric measurements (Kinra et al., 2014). SLI is a household level asset-based scale (covering quality of housing, including type of fuel use, and ownership of land and durable goods), ranging from 0 to 67, developed for use in Indian survey (International Institute for Population Sciences (IIPS) and ORC Macro, 2000; Kinra et al., 2014). Weight (measured to the nearest 0.1 kg by digital SECA machine) and standing height (nearest 1 mm by Leicester plastic stadiometer, Chasmors, UK) were measured twice; we used the average of the two values. Body mass index (BMI) was calculated as weight (kg)/height (m^2). We defined BMI categories using cut-off values adapted to the Indian population (Underweight as $<$18.0 kg/m^2, normal weight as 18.0–22.9 kg/m^2, overweight as 23.0–24.9 kg/m^2 and obese as $\geq$25 kg/m^2)(Misra et al., 2009).

2.3. Outcome

The primary outcome was forced expiratory volume in the first second (FEV_1), followed by the forced vital capacity (FVC). In general, FEV_1 is an indicator of airway resistance and FVC is an indicator of lung capacity. Lung function was measured following the ATS/ERS guidelines (Miller et al., 2005) with a standard operating procedure protocol with the Card-Guard Spiro-Pro (Card Guard) device. All measures were taken without bronchodilator administration and aimed to obtain three acceptable blows from a maximum of eight blows. We developed a shiny app to revise and classify all measured blows (Milà and Ranzani, 2022). First, we applied the acceptability criteria to every blow evaluating both volume-time and flow-volume curves (Miller et al., 2005). Second, we selected the best three curves that fulfilled the acceptability criteria. Following guidelines (Miller et al., 2005), we used both FEV_1 and FVC when acceptability criteria were present for both measures, and FEV_1 when the acceptability criteria was only present for FEV_1. The final value of FEV_1 and FVC was selected from the maximum value obtained among the three best ranked blows of those which fulfilled acceptability criteria. We considered the repeatability criteria as difference $\leq$150 ml for the two largest FEV_1 and FVC values. The lung function assessment is shown in Fig. 1. We did not evaluate z-scores using the Global Lung Function Initiative (GLI) equations as secondary outcomes because they are not suitable for the Indian subcontinent (Quanjer et al., 2012).

2.4. Exposure assessment

We estimated annual ambient concentration of $PM_{2.5}$ at residence using a land-use regression (LUR) model developed for the study area (Sanchez et al., 2018). The measurements and modelling approach have been detailed elsewhere (Sanchez et al., 2018; Tonne et al., 2017). The $PM_{2.5}$ LUR model included tree coverage, night-time light intensity, longitude and normalized difference vegetation index predictors, and explained a 58% (mean adjusted R^2) of the variation in measured $PM_{2.5}$ (Sanchez et al., 2018).

HAP was defined with self-reported data on cooking fuel. We derived a binary variable accounting for biomass fuel use (crop residues/dung/wood/kerosene/oil), compared with participants using clean fuel (gas/electricity). Additionally, we derived a three category variable, expanding the group using biomass fuel use in two groups: those with an unvented and those with a vented to the outside biomass-fuelled stove (Aung et al., 2016; Grieshop et al., 2017; Islam et al., 2021; Ranzani et al., 2020).

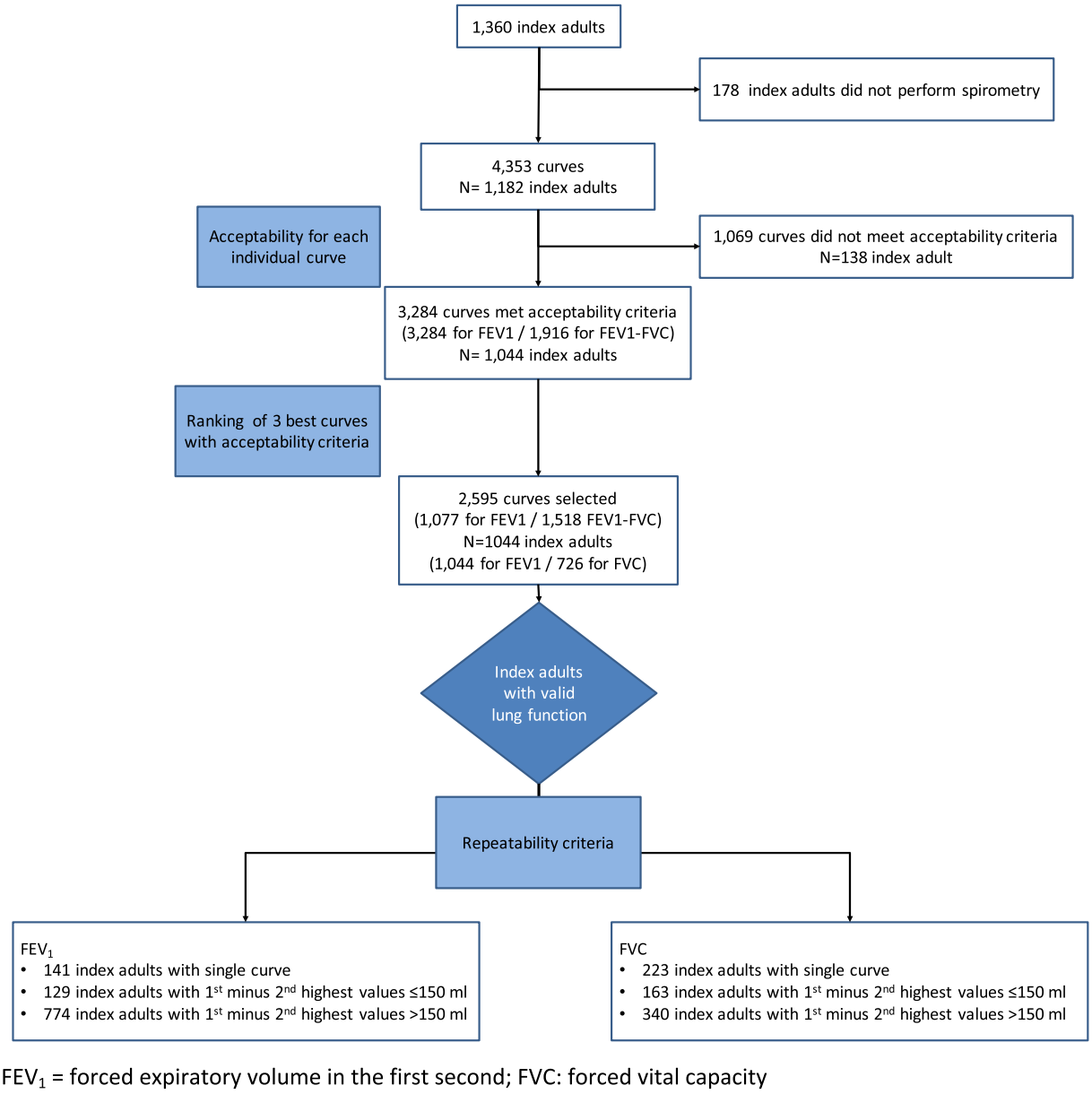

Fig. 1. Study flow chart. FEV_1 = forced expiratory volume in the first second; FVC: forced vital capacity.

2.5. Data analysis

The statistical analysis plan was defined a priori and any deviation is labelled post-hoc. We evaluated the associations between ambient $PM_{2.5}$ and HAP with FEV_1 and FVC fitting linear mixed models, with random intercepts by village. Ambient $PM_{2.5}$ and HAP were co-adjusted simultaneously in all models. We used a "within-between" approach in the mixed models because of the modest within-village variability in ambient $PM_{2.5}$ and to account for confounding at the village level (Adar et al., 2010; Bafumi and Gelman, 2006; Ranzani et al., 2020). For this, we included the village-mean ambient $PM_{2.5}$ as a covariate of the model.

Each set of potential confounding factors were chosen accordingly to the literature, previous knowledge, and reported associations in the APCAPS population. We sequentially adjusted for confounders as follows: Model 1 was adjusted by age (linear term), height (restricted cubic spline with 3 knots) and sex. Model 2 was further adjusted by smoking status, environmental tobacco smoke and BMI. Model 3 included Model 2 terms plus occupation and education level. Model 4 included Model 3 terms plus Standard of Living Index, which incorporates an indicator of biomass fuel use included in the score and therefore is at risk of over-adjustment. Finally, to increase precision, Model 5 added to Model 4 two dummy indicators: whether the participant had upper respiratory infection symptom in the past three weeks and a three-category variable regarding spirometry quality (one curve available, $\geq$2 curves without repeatability criteria, and $\geq$2 curves with repeatability criteria). Inclusion of the dummy variable for spirometry quality was defined post-hoc. We tested continuous variables for non-linearity with restricted cubic splines and allowed for non-linearity using AIC criteria. Given the sex-based differences in mobility and cooking time observed in the APCAPS population (Milà et al., 2018), we tested an effect modification between each exposure and sex by adding an interaction term between the exposure and sex.

We used inverse probability weighting (IPW) to adjust for potential selection bias among those with a valid spirometry compared with the 1,360 potentially eligible participants (Ranzani et al., 2020; Seaman and White, 2013). IPW was constructed using a logistic regression model, including the exposures, all potential confounding factors and a village indicator. We derived IPWs for having a valid FEV_1 and for FVC

separately. The primary analysis used stabilized IPWs truncated at 1st and 99th percentile of weights distribution to deal with extreme weight values (Austin and Stuart, 2015).

We conducted four sensitivity analyses: 1) truncating at 5th and 95th percentile of weights distribution (Austin and Stuart, 2015); 2) analysing without IPW; 3) analysing including only non-smokers; 4) analysing the FEV_1 from those curves with both FEV_1 and FVC valid. Sensitivity analysis restricted to participants with repeatability criteria were not stable due to the sample size. We anticipated few missing values and pre-planned to conduct a complete case-analysis. All analyses were conducted with R-4.0.3, with the packages *tidyverse* (Wickham et al., 2019), *lme4* (Bates et al., 2015), and *sjstats* (Lüdecke, 2018).

3. Results

3.1. Study population

There were 1,044 participants with a valid lung function measurement (1,044 (77%) for FEV_1 and 726 (53%) for FVC) (Fig. 1). The characteristics of participants with and without a valid lung function measurement are shown in eTable-1 and eTable-2. Overall, participants without a valid lung function measurement were more likely to be female, underweight, and unemployed compared to those with a valid measurement.

The mean age was 22.8 (SD = 1.2) years (p25-p75: 22–24, range 20–26 years). 327 participants were female (31.3%). Overall, one third of participants were classified as underweight and 40.3% were currently exposed to environmental tobacco smoke. Only males reported smoking tobacco, representing 9% of all participants and 13.1% of males. The mean age starting tobacco smoking was 19.4 (SD = 3.2) years. Among all participants, 43% were unemployed, and females (65.1%) were more likely to be unemployed compared to males (32.9%). Additional characteristics are described in Table 1.

The mean $PM_{2.5}$ exposure was 32.9 (SD = 2.8) $\mu g/m^3$ (range 24.4–38.1 $\mu g/m^3$), and comparable between females and males. Most participants reported use of biomass as cooking fuel (75.9%) and 21.8% of participants reported use of biomass as cooking fuel in an unvented stove (Table 1).

3.2. Association between ambient $PM_{2.5}$ and lung function

The final population included in regression analyses was 987/1,044 (95%) for FEV_1 and 682/726 (94%) for FVC, after excluding missing values in covariates. The association between 1 $\mu g/m^3$ increase in $PM_{2.5}$ and FEV_1 was −44 ml (95% CI, −106 to 18) in the minimally adjusted model and −27 ml (95% CI, −89 to 34) in the fully adjusted model, while the association between 1 $\mu g/m^3$ increase in $PM_{2.5}$ and FVC was −5 ml (95% CI, −91 to 80) in the minimally adjusted model and −8 ml (95% CI, −93 to 76) in the fully adjusted model (Table 2). Results were similar when HAP was modelled as three categories rather than two (eTable-3).

3.3. Association between household air pollution and lung function

HAP was associated with decreased FEV_1 in the minimally adjusted model (−93 ml, 95% CI, −188 to 3) and in the fully adjusted model (−112 ml, 95% CI, −211 to −13); this association was of greater magnitude for those with unvented stove (fully adjusted model: −93 ml, 95% CI, −196 to 10 for those with vented stove, and −158 ml, 95% CI, −279 to −36 for those with unvented stove) (Fig. 2, eTable-4 and eTable-5). The same pattern was observed in the association between HAP and FVC: in the fully adjusted model, HAP was associated with −142 ml (95% CI, −285 to 0); when considering the ventilation, it was −114 ml (95% CI, −263 to 35) for those with a vented stove and −211 ml (95% CI, −386 to −36) for those with an unvented stove (Fig. 2, eTable-4 and eTable-5).

Table 1
General characteristics, outcomes and exposure of the population stratified by sex.

	Category	Overall	Female	Male
n		1044	327	717
Age, years	Mean (SD)	22.8 (1.2)	22.7 (1.3)	22.9 (1.2)
Sex, n (%)	Female	327 (31.3)	327 (100.0)	–
Height, m	Mean (SD)	1.63 (0.09)	1.53 (0.06)	1.67 (0.06)
Body-mass index, kg/m^2	Mean (SD)	20.5 (3.2)	20.1 (3.3)	20.7 (3.2)
Body-mass index categories (kg/m^2), n (%)	Underweight (<18.0)	312 (29.9)	116 (35.5)	196 (27.3)
	Normal weight (18.0–22.9)	516 (49.4)	158 (48.3)	358 (49.9)
	Overweight (23.0–24.9)	122 (11.7)	26 (8.0)	96 (13.4)
	Obese (25.0 -)	94 (9.0)	27 (8.3)	67 (9.3)
Environmental tobacco smoke, n (%)	Yes	421 (40.3)	98 (30.0)	323 (45.0)
Tobacco smoking, n (%)	Yes	94 (9.0)	0 (0.0)	94 (13.1)
Starting age of tobacco smoking, years	Mean (SD)	19.4 (3.2)	–	19.4 (3.2)
Physical activity (METs), n (%)	Sedentary or light active (<1.70)	897 (87.4)	270 (84.6)	627 (88.7)
	Active or moderately active (1.70–1.99)	106 (10.3)	39 (12.2)	67 (9.5)
	Vigorously active (>2)	23 (2.2)	10 (3.1)	13 (1.8)
Education, n (%)	No formal education	75 (7.2)	35 (10.7)	40 (5.6)
	Primary (1–4 years)	129 (12.4)	54 (16.5)	75 (10.5)
	Secondary (5–12 years)	616 (59.0)	179 (54.7)	437 (60.9)
	Beyond secondary (>12 years)	224 (21.5)	59 (18.0)	165 (23.0)
Occupation, n (%)	Unemployed	449 (43.0)	213 (65.1)	236 (32.9)
	Unskilled manual	177 (17.0)	56 (17.1)	121 (16.9)
	Skilled manual	312 (29.9)	34 (10.4)	278 (38.8)
	Non-manual	106 (10.2)	24 (7.3)	82 (11.4)
Standard of Living Index (points), n (%)	Low (0–14)	15 (1.4)	5 (1.5)	10 (1.4)
	Medium (15–24)	240 (23.1)	80 (24.7)	160 (22.4)
	High (25–67)	783 (75.4)	239 (73.8)	544 (76.2)
Outcomes				
FEV_1, L	Mean (SD)	3.27 (0.92)	2.48 (0.55)	3.63 (0.82)
FVC, L	Mean (SD)	4.17 (1.07)	3.19 (0.62)	4.54 (0.97)
Exposures				
$PM_{2.5}$ ($\mu g/m^3$)*	Mean (SD)	32.9 (2.8)	32.9 (2.8)	32.8 (2.7)
Fuel use for cooking, n (%)	No biomass	251 (24.0)	82 (25.1)	169 (23.6)
	Biomass, ventilated stove	565 (54.1)	195 (59.6)	370 (51.6)
	Biomass, no ventilated stove	228 (21.8)	50 (15.3)	178 (24.8)

* Missing values were 35 (3.4%) for PM2.5, 18 (1.7%) for physical activity and 6 (0.6%) for Standard of Living Index FEV_1 = forced expiratory volume in the first second; FVC: forced vital capacity; PM 2.5 = particulate matter with an aerodynamic diameter of 2.5 μm or less.

Table 2
Association of within-village variation in $PM_{2.5}$ with lung function in young adults in a peri-urban community in India.

Model	Exposure	Overall
Outcome: FEV_1 (ml)		N = 987 Mean difference in FEV_1 (95% CI), ml
Ambient $PM_{2.5}$		
Model 1 (basic adjustment)[a]	$PM_{2.5}$ (1 μg/m^3)	−44 (-106 to 19)
Model 2 (Model 1 + health behaviours and BMI)[b]	$PM_{2.5}$ (1 μg/m^3)	−43 (-105 to 19)
Model 3 (Model 2 + occupation and education)[c]	$PM_{2.5}$ (1 μg/m^3)	−27 (-89 to 35)
Model 4 (Model 3 + socioeconomic index)[d]	$PM_{2.5}$ (1 μg/m^3)	−27 (-89 to 35)
Model 5 (Full adjustment)[e]	$PM_{2.5}$ (1 μg/m^3)	−27 (-89 to 35)
Outcome: FVC (ml)		N = 682 Mean difference in FVC (95% CI), ml
Ambient $PM_{2.5}$		
Model 1 (basic adjustment)[a]	$PM_{2.5}$ (1 μg/m^3)	−5 (-91 to 80)
Model 2 (Model 1 + health behaviours and BMI)[b]	$PM_{2.5}$ (1 μg/m^3)	−14 (-99 to 70)
Model 3 (Model 2 + occupation and education)[c]	$PM_{2.5}$ (1 μg/m^3)	−9 (-93 to 76)
Model 4 (Model 3 + socioeconomic index)[d]	$PM_{2.5}$ (1 μg/m^3)	−7 (-92 to 77)
Model 5 (Full adjustment)[e]	$PM_{2.5}$ (1 μg/m^3)	−8 (-93 to 76)

Analysis conducted using a linear mixed model accounting for within-between effects for $PM_{2.5}$, with correction for selection bias through inverse probability weighting. CI = confidence interval; FEV_1 = forced expiratory volume in the first second; FVC: forced vital capacity; PM 2.5 = particulate matter with an aerodynamic diameter of 2.5 μm or less.

[a] Model 1 was adjusted by age (modelled with linear term), height (modelled with restricted cubic spline with 3 knots), sex and $PM_{2.5}$ and biomass use (yes/no).

[b] Model 2: Model 1 + smoking status, environmental tobacco smoke and body-mass index.

[c] Model 3: Model 2 + occupation and education.

[d] Model 4: Model 3 + Standard of Living Index.

[e] Model 5: Model 4 + symptom of upper respiratory infection in the last three weeks and FEV_1/FVC quality indicator.

3.4. Effect modification by sex

We did not observe effect modification by sex for ambient $PM_{2.5}$, while the association for HAP was of greater magnitude in male than female (eTable-6).

3.5. Sensitivity analyses

The four sensitivity analyses yielded comparable estimates to the main analysis (Fig. 3 and eTable-7), in general with wider confidence intervals for those analyses with lower sample size.

4. Discussion

4.1. Main findings

We observed an association between air pollution and decreased no-bronchodilator lung function in young adults residing in a peri-urban area of South India. Long-term ambient air pollution measured by annual average $PM_{2.5}$ was associated with decreased FEV_1, but with imprecision, while household air pollution measured by biomass fuel use was consistently associated with decreased FEV_1 and FVC, particularly in those cooking with biomass in unventilated stoves.

Our estimates for ambient $PM_{2.5}$ and FEV_1 are broadly consistent with those from previous studies. A systematic review and meta-analysis published in 2019 found that, for healthy adults, the pooled estimate from four studies (n = 56,125) for the decrease on FEV_1 was −71.4 ml (95% CI, −134.5 to −8.2) per 10 μg/m^3 increase of PM_{10} (Edginton et al., 2019). Another cross-sectional analysis comprising several cohorts of Europe (n = 7,615) observed a decrease of −44.56 ml (95% CI, −85.36 to −3.76) per 10 μg/m^3 increase of PM_{10}, however observed non-statistically significant associations for $PM_{2.5}$ (-21.14 ml, 95% CI, −56.37 to 14.08 for FEV_1 and −36.39, 95% CI, −83.29 to 10.50 for FVC, per 5 μg/m^3 increase) and other PM diameters (Adam et al., 2015). A recent large study (n = 285,046) from Taiwan observed consistent decreases in FEV_1 and FVC for $PM_{2.5}$ in a relatively high exposure range (average $PM_{2.5}$ of 26.74 ± 7.76 μg/m^3) (Guo et al., 2018). Similarly, another large study in UK (UK-Biobank, n = 278,228), observed consistent negative associations between $PM_{2.5}$ and FEV_1 or FVC, with a greater effect in males (Doiron et al., 2019). Among adolescents, previous studies have reported no association between $PM_{2.5}$ and FEV_1 or FVC in the general population (Cai et al., 2020; Fuertes et al., 2015), but negative associations have been reported in some subgroups such as males for $PM_{2.5}$, or asthmatics for other pollutants (Fuertes et al., 2015). We observed null associations for ambient $PM_{2.5}$ and FVC. The discrepancy of the negative association only with FEV_1 and not FVC has also been observed in another study of adolescents (~16 years) in a cohort in Netherlands (Milanzi et al., 2018), which observed stronger effects in boys.

There is some evidence for the association of HAP and chronic respiratory diseases from both LMIC and high-income countries (Orozco-Levi et al., 2006; van Gemert et al., 2015). However, few studies have investigated the effect of HAP on lung function measures in the general population (Amaral et al., 2018; Arku et al., 2020; Dave et al., 2017; Kashyap et al., 2020; Sood et al., 2018). The BOLD study analysed data on adults (mean age 50–60 years) in several countries, and reported no associations between solid biomass fuel and FEV_1 or FVC overall or in subgroups by sex, smoking and country income status (Amaral et al., 2018). Results from another multinational study (PURE) based on adults (35–70 years) indicated that the use of kerosene for cooking was negatively associated with FEV_1 (−46.27 ml, 95% CI, −80.47 to −12.06) and FVC (−54.67 ml, 95% CI, −93.59 to −15.75) compared with clean fuel (Arku et al., 2020). Associations were greater in magnitude for males and among participants from India (−127.27 ml, 95% CI, −171.33 to −83.2 for FEV_1). Finally, a cross-sectional study in India including adults (mean age 49.4 years) observed that those using biomass fuel for cooking had −70 ml (95% CI, −111 to −30) smaller FEV_1 compared with those with clean fuel. This difference was mainly observed in males (−145 ml, 95% CI, −210 to −79), while results were inconclusive for females (−13 ml, 95% CI, −63 to 37) (Dave et al., 2017). The associations were imprecise for FVC (−10 ml, 95% CI, −70 to 50 overall, −56 ml, 95% CI, −138 to 26 for males) and there was no effect modification by cooking in a living space or enclosed kitchen. Although we did not find any study evaluating HAP and lung function in young adults, our estimates are broadly comparable with estimates observed among older adults. Our observed associations were mainly driven by participants using unvented stoves, providing additional support that exposure measured by questionnaire data on primary cooking fuel is reflecting exposure to combustion by products and that the observed association cannot be fully explained by residual confounding by socio-economic factors correlated with cooking fuel (Mortimer et al., 2022).

Several pathways may be involved in the biological mechanism linking ambient $PM_{2.5}$ and HAP with lung function, including chronic inflammation, increased airway resistance, and changes in the lung microbiome (Pinkerton et al., 2019; Raju et al., 2020; Sood et al., 2018; Stapleton et al., 2020). There is also published evidence for delayed or decreased lung function growth in children exposed to air pollution (Aithal et al., 2021; Cai et al., 2020; Götschi et al., 2008; Schultz et al., 2016), and potential for improved lung function in children following reductions in ambient air pollution (Gauderman et al., 2004). However,

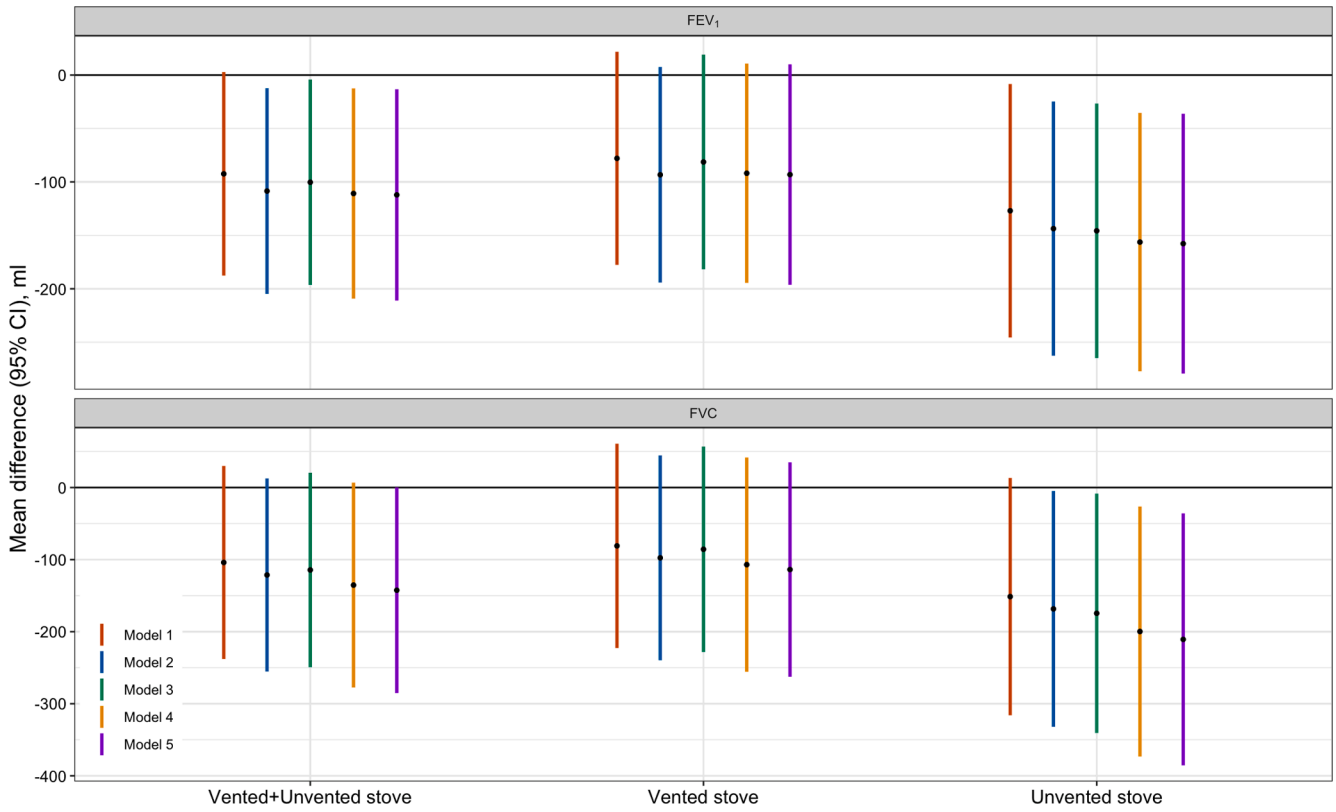

Fig. 2. Association between household air pollution and lung function in young adults in a peri-urban community in India. Analysis conducted using a linear mixed model accounting for within-between effects for PM2.5, with correction for selection bias through inverse probability weighting. CI = confidence interval; FEV1 = forced expiratory volume in the first second; FVC: forced vital capacity; PM 2.5 = particulate matter with an aerodynamic diameter of 2.5 μm or less. Model 1 was adjusted by age (modelled with linear term), height (modelled with restricted cubic spline with 3 knots), sex and PM2.5 and biomass use (yes/no and three categories). Model 2: Model 1 + smoking status, environmental tobacco smoke and body-mass index. Model 3: Model 2 + occupation and education. Model 4: Model 3 + Standard of Living Index. Model 5: Model 4 + symptom of upper respiratory infection in the last three weeks and FEV1/FVC quality indicator.

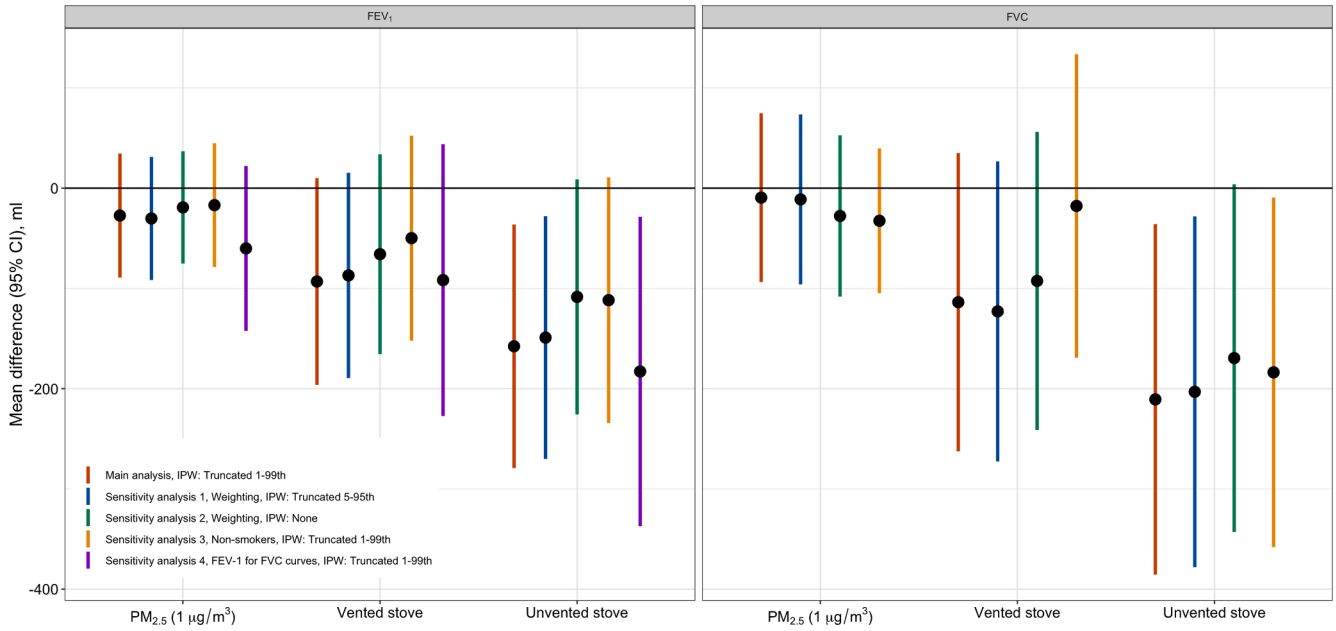

Fig. 3. Sensitivity analyses for the association between ambient $PM_{2.5}$ and household air pollution and lung function. Sensitivity analyses conducted using a linear mixed model accounting for within-between effects for PM2.5. CI = confidence interval; FEV1 = forced expiratory volume in the first second; FVC: forced vital capacity; PM 2.5 = particulate matter with an aerodynamic diameter of 2.5 μm or less. Results from Model 5: adjusted by age (modelled with linear term), height (modelled with restricted cubic spline with 3 knots), sex and PM2.5, biomass use (3 categories), smoking status, environmental tobacco smoke and body-mass index, occupation, education, Standard of Living Index, symptom of upper respiratory infection in the last three weeks and FEV1/FVC quality indicator.

the literature is more limited for adolescents, and available findings are not as consistent as they are for younger children. Additionally, it is not clear whether there is a sex-specific impact of air pollution (Cai et al., 2020; Fuertes et al., 2015; Götschi et al., 2008; Milanzi et al., 2018), or whether ambient PM is more relevant for FEV_1 (increased airway resistance/obstructive) compared with FVC (lung capacity) (Milanzi et al., 2018).

Our study is one of the first evaluating air pollution and lung function of young adults directly following their lung peak growth. The attained maximum lung function is one of the main determinants of lung function decline trajectory and incidence of respiratory diseases and premature death (Agustí et al., 2017; Rennard and Drummond, 2015). Another strength of our study is that we evaluated co-exposure to ambient $PM_{2.5}$ and HAP in a population-based study in a low-and-middle income country, where exposure to high levels of ambient $PM_{2.5}$ and household air pollution are prevalent. Additionally, we adjusted for occupation and other socioeconomic indicators, which are important confounders for HAP (Arku et al., 2020; Mortimer et al., 2022), and results were consistent across multiple different sensitivity analyses.

Limitations of our study include the following. First, this is a cross-sectional analysis thus we cannot evaluate the impact of air pollution exposure on lung growth and the influence of different exposure windows. Second, HAP was evaluated using a categorical exposure, consequently, we were not able to estimate exposure-response relationships and accurately evaluate the personal exposure generated from HAP. Although we co-adjusted for ambient $PM_{2.5}$ and a HAP indicator, isolating the effect of each on lung function is not straightforward since some ambient $PM_{2.5}$ results from local biomass burning for household energy. Previous studies from our group showed that about 8–12% of ambient $PM_{2.5}$ in the study area resulted from local sources (Kumar et al., 2018). Third, there was a considerable proportion of invalid spirometry curves, more commonly for females and for participants with low socioeconomic status, which is a common challenge for evaluating lung function in population-based studies in LMICs (Masekela et al., 2018; Meghji et al., 2021). Additionally, we have a higher proportion of males compared to females (38.6%) in the eligible population of index adults of third APCAPS follow-up and females were much less likely to have a valid FEV_1 measure or to meet acceptability/repeatability criteria compared with males. Therefore, we could have selection bias and lack of representative of females on our analysis, which we tried to minimize using IPW. The use of a categorical variable as a proxy of HAP, likely with differential measurement error between sex associated with potential selection bias, might explain the inconclusive magnitude of the association between HAP and lung function in females. Additional studies should plan actions to mitigate the likely lack of representativeness of females in lung function studies in LMICs (Masekela et al., 2018; Meghji et al., 2021). Fourth, we did not have post-bronchodilator lung function measurements, which are needed to evaluate obstructive respiratory diseases prevalence. Post-bronchodilator lung function measurements could also reduce the influence of short-term environmental exposures on FEV_1 measurements (Ierodiakonou et al., 2016; Mölter et al., 2013), potentially improving the precision of our estimates of interest (i.e. associations between long-term ambient $PM_{2.5}$ and HAP exposures and lung function).

5. Conclusions

We observed negative associations between ambient $PM_{2.5}$ and HAP and lung function in young adults who had recently attained their maximum lung function. Because maximum attained lung function is a main determinant of lung function in later life, aiming to reduce the levels of exposure to ambient $PM_{2.5}$ and HAP might be an effective way to improve lung function in adulthood.

Ethics approval.

APCAPS was. approved by the London School of Hygiene & Tropical Medicine (London, UK) and the National Institute of Nutrition (NIN) (Hyderabad, India). CHAI was approved by the Ethics Committees of Parc de Salut MAR (Barcelona, Spain), the Indian Institute of Public Health (Hyderabad, India), and the NIN. Signed consent forms were obtained from all participants.

Role of the funding sources

The funders had no role in the design and conduct of the study; in the collection, analysis, and interpretation of the data; in the preparation, review, or approval of the manuscript; or in the decision to submit the manuscript for publication. All authors were responsible for the decision to submit for publication.

Data sharing

Data related to the APCAPS cohort is available to researchers through a brief application to the cohort's Steering Group (form available from APCAPS website, https://apcaps.lshtm.ac.uk, and submitted to email: apcaps@iiphh.org).

CRediT authorship contribution statement

Otavio T. Ranzani: Conceptualization, Data curation, Formal analysis, Methodology, Software, Validation, Visualization, Writing – original draft. **Santhi Bhogadi:** Data curation, Project administration, Writing – review & editing. **Carles Milà:** Methodology, Software, Visualization, Writing – review & editing. **Bharati Kulkarni:** Data curation, Writing – review & editing. **Kalpana Balakrishnan:** Data curation, Writing – review & editing. **Sankar Sambandam:** Data curation, Writing – review & editing. **Judith Garcia-Aymerich:** Methodology, Supervision, Writing – review & editing. **Julian D. Marshall:** Data curation, Writing – review & editing. **Sanjay Kinra:** Data curation, Methodology, Funding acquisition, Project administration, Writing – review & editing. **Cathryn Tonne:** Conceptualization, Data curation, Funding acquisition, Methodology, Project administration, Resources, Supervision, Validation, Writing – review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2022.107290.

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