How Do Household Energy Transitions Work?

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1 Introduction

China is deploying an ambitious policy to transition up to 70% of households in northern China to clean space heating, including a large-scale roll out across rural and peri-urban Beijing. To meet this target the Beijing municipal government announced a two-pronged program that designates coal-restricted areas and simultaneously offers subsidies to night-time electricity rates and for the purchase and installation of electric-powered, air-source heat pumps to replace traditional coal-heating stoves. The policy was piloted in 2015 and, starting in 2016, was rolled out on a village-by-village basis; however there is uncertainty as to when villages will receive the program. The variability in when the policy is applied to each village allows us to treat the roll-out of the program as a quasi-randomized intervention. Households may also be differentially affected by this program due to factors such as financial constraints, preferences and social capital, and there is uncertainty about whether and how this intervention may affect indoor and outdoor air pollution, as well as heating behaviors and health outcomes.

2 Background

2.1 Context for the policy

Beijing has a temperate continental monsoon climate characterized by cold, dry winters and hot, humid summers. Access to central heating is limited to urban areas and households in most rural and peri-urban areas of Beijing historically heated their homes using mostly coal and sometimes biomass-fueled heaters or kangs (a traditional Chinese combined cooking and heating stove). Household coal burning was a major contributor to indoor and outdoor air pollution in northern China, especially in winter. Prior to 2016, coal fuel was used to meet over 80% of northern China's space heating demand (Dispersed Coal Management Research Group 2023). At that time, household coal-fuelled heaters burned approximately half of the over 400 million tons of coal used for space heating (Group 2016) and contributed to ~30% of northern China's wintertime air pollution. In 2013, exposure to ambient fine particulate matter from coal combustion - from industry, electricity,

and domestic sources - was the largest estimated contributor to population exposure to $PM_{2.5}$ and contributed to an estimated 366,000 premature deaths annually in China (Group 2016).

Replacing household coal stoves with clean heating alternatives was considered a potentially impactful intervention to reduce outdoor $PM_{2.5}$ across the region and mitigate its health impacts. A number of clean heating options including electric heat pumps, gas heaters, and electric resistance heaters with thermal storage were widely promoted by the Chinese government (Dispersed Coal Management Research Group 2023). By 2021, over 36 million households in northern China were treated by the policy and an estimated 21 million additional households expected to be treated by 2025. Whether this large-scale energy policy yielded air quality and health benefits remains a critical and unresolved question.

2.2 Prior evidence of household energy interventions and air pollution

Household energy interventions, mostly cooking-related, that replace solid fuel stoves with cleaner-burning alternatives have been implemented and studied extensively in countries including China over the past several decades. While their introduction is expected to reduce air pollution emissions and subsequent exposures, there is still no consensus about their effectiveness in achieving health-relevant air pollution reductions in real-world settings (Quansah et al. 2017). In particular, the effectiveness of large-scale household energy programs like China's Coal Ban and Heat Pump (CBHP) subsidy program Clean Heating Plan has been rarely empirically investigated, especially at sub-city spatial resolution. In Ireland, county-level residential coal bans in the 1990s were associated with 40-70% decreases in black smoke concentrations in ban-affected areas (Dockery et al. 2013). In Australia, a wood-burning stove exchange lower daily wintertime PM10 from 44 to 27 µg/m3 (Johnston et al. 2013), and clean energy policies in New Zealand were associated with 11-36% reductions in winter PM10 (Scott and Scarrott 2011). The few evaluations of the Clean Heating Plan observed small decreases in outdoor $PM_{2.5}$ (-7 to -2.4 µg/m3) in municipalities or prefectures in the policy compared with neighboring areas not affected by the policy (Niu et al. 2024; Song et al. 2023; Tan et al. 2023; Yu et al. 2021), and a recent modeling study estimated 36% lower personal exposure to $PM_{2.5}$ based on household-reported changes in fuel use (Meng et al. 2023). However, none of these studies included field-based measurements of air pollution or personal exposures, which are known to differ considerably from modeled estimates (Thompson et al. 2019), and few accounted for secular changes in air quality over time, limiting any conclusions about the air quality benefits of the Clean Heating Plan.

2.3 Prior evidence on clean energy interventions and cardiovascular outcomes

Most previous health assessments of household energy interventions have focused on cookstoves rather than heating. Randomized trials of less polluting cookstoves generally indicate a potential cardiovascular benefit. In older Guatemalan women, a chimney stove intervention lowered exposure

to air pollution and reduced the occurrence of nonspecific ST-segment depression. That same study and other trials in Nigeria and Ghana observed blood pressure reductions (range: -3.7 to -1.3mmHg) in women assigned to gas, ethanol, or improved combustion biomass stoves, and are supported by non-randomized, controlled intervention studies in Nicaragua and Bolivia (blood pressure reductions from -5.9 to -5.5mmHg). A recent multi-country randomized trial did not observe a protective effect of gas stoves on gestational blood pressure despite large reductions in air pollution, though the participants were younger (mean age: 25y) than in intervention studies showing a blood pressure benefit (mean age range: 28 to 53y).

The few population-based evaluations of household energy policies also indicate a cardio-respiratory benefit. Residential wood-burning bans were associated with reductions in cardiovascular hospitalizations (-7%) in California and with reduced cardiovascular (-17.9%) and respiratory (-22.8%) mortality in Australia, though neither study fully controlled for secular improvements in health. Most relevant to our study are two quasi-experimental assessments of coal replacement policies. In Ireland, reductions in respiratory not but cardiovascular mortality were observed following a coal ban. A multi-city study of Chinese adults in cities where the Clean Heating Policy was piloted compared with adults in cities not in the pilot observed small decreases in chronic lung diseases (-3.0 to -1.1%) but no change in physician-diagnosed cardiovascular diseases, potentially due to the short (one-year) post-policy evaluation period or confounding by other unmeasured city-wide air quality or health-related policies.

2.4 Assessing dynamic and heterogeneous treatment effects

Since 2015, thousands of villages across Beijing and northern China entered the policy prior to the start of the heating season each year. Given the many behavioral, social, or economic factors that might affect both new heater use and coal stove suspension (e.g., energy prices and availability, wintertime temperature, COVID-19 pandemic, user preferences), it is possible that the effect of the policy on air pollution and health may be dynamic over time and/or heterogeneous across treatment cohorts. Thus, it may be important to study both the overall and group-time effects of the policy.

2.5 Evaluating the mechanisms through which policies may affect health outcomes.

With several notable exceptions, decades of household energy intervention studies indicating limited or even no benefit to air quality or health very clearly demonstrate that intervention is not simple when studying an exposure that is as central to daily life as household energy use (add refs). Household energy intervention and policies, particularly those implemented at the household- or village-scales, can produce multiple behavioral, environmental, and health-related changes, making it important to investigate the mechanisms through which such policies exert their health impacts (Dominici et al. 2014). The health benefits achievable with transition from traditional coal stoves to

a new electric home heating system, for example, may be influenced by factors including outdoor air quality (Lai 2019), the desirability and usage patterns of new and traditional stoves (Ezzati and Baumgartner 2017) indoor temperature (Lewington et al. 2012), or behaviors including physical activity (Lindemann et al. 2017). Only recently were these mediating factors considered in assessments of household energy interventions and health, and rarely in any comprehensive way (Rosenthal et al. 2018). Understanding these mechanisms can provide valuable scientific insight into the success (or failure) of clean energy policies like the Clean Heating Policy in meeting its air quality and health goals, and may answer questions that can inform the design of more effective future energy interventions. For example, is there successful uptake of the intervention or policy? Does the policy lead to heating behavior changes that result in colder homes and thus offsets any cardiovascular-enhancing effects of improved air quality? Answers to these questions are facilitated by the analysis of mediating pathways.

3 Specific Aims and Overarching Approach

This study used three data collection campaigns in winter 2018/19, winter 2019/20, and winter 2021/22, as well as a partial campaign in winter 2020/21 to advance the following aims:

1. Estimate how much of the policy's overall effect on health, including respiratory symptoms and cardiovascular outcomes (blood pressure, central hemodynamics, blood inflammatory and oxidative stress markers), can be attributed to its impact on changes in $PM_{2.5}$;

2. Quantify the impact of the policy on outdoor air quality and personal air pollution exposures, and specifically the source contribution from household coal burning (Previously Aim 3);

3. Quantify the contribution of changes in the chemical composition of $PM_{2.5}$ from different sources to the overall effect on health outcomes (Previously Aim 2).

4 Study Design and Methods

4.1 Location, context, and recruitment

In December 2018 we recruited 50 villages across 4 administrative districts (Fangshan, Huairou, Mentougou, and Miyun) in the Beijing Municipality Region in northern China. Villages were selected on the basis of their residents primarily using household coal stoves for heating, and because roughly half of the villages were expected to enter into the policy over the course of our study. We used local guides in each village to help determine a roster of households that were not vacant during the winter months, from which we randomly selected households to recruit for participation.

We recruited approximately 20 households in each village and randomly selected one eligible person from each household to participate. Participants were eligible to participate if they were over 40 years old, lived in the study villages, were not planning to move out of the village in the next year, and were not on current immunotherapy or treatment with corticosteroids. Research staff introduced the study and its measurements to an eligible person in each household and answered any questions related to the study. All participants provided written informed consent prior to joining the study. The study protocols were approved by research ethics boards at Peking University (IRB00001052-18090) and McGill University (A08-E53-18B).

4.2 Data Collection Overview

Trained staff traveled to participants' homes to conduct tablet-based household and individual questionnaires, measure their blood pressure, and place air pollution and temperature monitors in their homes. Anthropometrics (height, weight, and waist circumference), measurement of airway inflammation, and whole blood samples were obtained no more than a month later at a village clinic in the first and second campaigns. In the fourth campaign, which occurred during the COVID-19 pandemic, anthropometric measurements and airway inflammation were assessed in participant homes to avoid group contact and blood samples were not collected.

4.2.1 Air Pollution

4.2.1.1 Outdoor air pollution

In each village, two sensors were set up to measure community $PM_{2.5}$ at different locations in each village. One sensor was placed near the center of the village, and the other was placed no less than 500m away from the centrally-located sensor. Sensors were placed at least 1.5m above the ground and in a location without a visible point source of $PM_{2.5}$.

We collected filter-based community $PM_{2.5}$ samples to calibrate the sensor-based $PM_{2.5}$ measurements. Ultrasonic Personal Aerosol Samplers (UPAS, Access Sensor Technologies, Fort Collins, CO, USA) were used to collect filter-based $PM_{2.5}$ samples with a flow rate of 1.0 L/min (Volckens et al. 2017). Samplers housed 37mm PTFE filters (VWR, 2.0- m pore size) and were equipped with a cyclone inlet with a 2.5 m cut point designed to perform under the sampling flow rate. For community outdoor measurements, a UPAS was co-located with each $PM_{2.5}$ sensor in each village in rotation. Every week, the used filters were removed and replaced with a new filter. In total, 126, 371, and 289 filter-based, community outdoor $PM_{2.5}$ samples were collected in seasons 1, 2, and 4, respectively. Field blank filters were collected at a rate of ~10%, subject to the same field conditions as samples.

For $PM_{2.5}$ sensor calibration and quality control, all PM sensors were co-located with a reference-grade $PM_{2.5}$ instrument (Model 5030 Synchronized Hybrid Ambient Realtime Particulate (SHARP)

Table 1: Household recruitment for overall and indoor air quality measurements.

Sample	Overall			Indoor		
	Season 1	Season 2	Season 4	Season 1	Season 2	Season 4
New recruitment	977	0	196	300	68	52
Households from Season 1	\	\	866	0	780	0
Households from Season 2	\	\	\	\	162	248
Total recruitment	977	0	1062	300	1010	300

Monitor, Thermo Fisher Scientific, United States) on the rooftop of a building at Peking University campus for 7 to 10 days before and after each field campaign. Sensor-measured $PM_{2.5}$ concentrations were highly correlated with those measured by the SHARP (Spearman correlation coefficients (rho) of 0.95 and 0.82 in pre- and post-calibration, respectively).

We established linear regression models between the filter-based $PM_{2.5}$ mass concentrations (i.e., the reference concentrations) and the sensor-based $PM_{2.5}$ concentrations averaged over the same sampling period as the filter-based samples. The slopes of the models were used as the adjustment factors for the sensor-based $PM_{2.5}$ concentrations. Separate regression models were conducted for indoor and outdoor sensors given the sensitivity of the sensors to relative humidity, temperature, and particle sources, which may differ for indoor versus outdoor conditions.

4.2.1.2 Indoor $PM_{2.5}$

In the second and fourth field seasons (i.e., Season 2 and Season 4), we randomly selected six households from the 20 recruited in each village to measure indoor concentrations of $PM_{2.5}$. In Season 4, we aimed to monitor indoor $PM_{2.5}$ in the same households where we measured indoor $PM_{2.5}$ in Season 2. If a household dropped out of the project or declined indoor $PM_{2.5}$ monitoring, we then recruited another household already enrolled in this study to measure indoor $PM_{2.5}$. In total, indoor measurements were conducted in 300 households in both Season 2 and Season 4 (Table 1).

Time-resolved indoor $PM_{2.5}$ concentrations were measured using the same commercially available sensor (PMS7003 Plantower, Zefan, Inc.) as was used for outdoor sensor-based $PM_{2.5}$ measurements and recorded $PM_{2.5}$ concentrations every 1 min. The sensor was placed on a table in a room where participants reported spending most of their time when awake, e.g., a living room or bedroom. Indoor $PM_{2.5}$ sensors were deployed between late November and mid January within field seasons (i.e., Season 2 and Season 4), depending on the village and household visit schedule. The measurement continued from the time of deployment until sensors were recollected from homes in late April.

We randomly selected three households from the six households in which we deployed $PM_{2.5}$ sensors to co-locate a filter-based $PM_{2.5}$ sampler with the $PM_{2.5}$ sensor. We collected a 24-h $PM_{2.5}$ filter sample at the first 24-h of indoor $PM_{2.5}$ sensor measurements. Filter-based $PM_{2.5}$ samples were collected using Ultrasonic Personal Aerosol Samplers (UPAS, Access Sensor Technologies) or Personal Exposure Monitors (PEMs, Apex Pro) operating with flow rates of 1.0 and 1.8 L/min, respectively. Both samplers housed 37 mm PTFE filters (VWR, 2.0- m pore size) and were equipped with a cyclone inlet with a 2.5 m cut point designed to perform under the corresponding sampling flow rate. After 24-h, the samplers were retrieved and loaded with new filters for measurements in other villages, once the previous sample filters were removed and stored for later analysis. In total, we successfully collected 149 and 148 indoor $PM_{2.5}$ filter samples in Seasons 2 and 4, respectively.

4.2.1.3 Personal exposure to $PM_{2.5}$ and black carbon

To measure personal exposure we used two types of samplers: Personal Exposure Monitors (PEMs, Apex Pro; Casella, UK) and Ultrasonic Personal Aerosol Samplers (UPAS, Access Sensor Technologies, Fort Collins, CO, USA). PEMs actively sampled air at a flow rate of 1.8 L/min, and UPAS sampled air at 1.0 L/min (Volckens et al. 2017). Both samplers housed 37 mm PTFE filters (VWR, 2.0- m pore size) and were equipped with a cyclone inlet with a 2.5 m cutpoint. Sampler flow rates were calibrated the night before deployment and also measured after the sampling period. Very few post-sampling measurements (<2%) deviated from the target flow rate by > +/-10%. Participants were instructed to wear a small waistpack (for the PEM and sampling pump) or an arm band or cross-body sling (for the UPAS) for 24 hours, which they could remove from their body and place within 2 meters while sleeping, sitting, or bathing. Field blanks for personal air pollution exposure measurements were collected at a rate of ~10% in each village. All filters were placed in individually labeled cases, sealed in plastic bags, and then transported to a field laboratory and immediately stored in a -20°C freezer. Following completion of the field sampling campaign, the samples and blanks were transported to Colorado State University, where they were stored in a -20°C freezer prior to $PM_{2.5}$ mass measurement and chemical analysis of PM.

All filters were placed in an environmentally-controlled equilibration chamber (21-22 °C, 30-34% relative humidity) for at least 24 hours before tare and gross weighing. Before each weight was taken, filters were discharged by a polonium-210 strip. Filters were weighed on a microbalance (Mettler Toledo Inc., XS3DU, USA) with 1- g resolution in triplicate or more, until the differences among three weights were less than 3 g. The average of three readings was used to determine filter mass, which was then blank-corrected using the median value of blank filters [3 g for UPAS-collected filters (53% of samples); 33 g for PEM-collected filters (47% of filter samples)], and PM2.5 concentrations were calculated by dividing the mass by the sampled air volume.

Filters were analyzed for black carbon (BC) using an optical transmissometer data acquisition system (SootScan^TM OT21 Optical Transmissometer; Magee Scientific, Berkeley, CA, USA). Light attenuation through each filter was measured before and after sampling in the field. To calculate BC mass, the difference between the pre- and post- light attenuation was converted to a mass

surface loading using the classical Magee mass absorption cross-sections of 16.6 m2/g for the 880 nm channel optical BC (Ahmed et al. 2009). BC concentrations were calculated by multiplying surface loadings by the sampled surface area of the filters (8.6 cm2 for UPAS-collected filters; 7.1 cm2 for PEM-collected filters), correcting for the field blank mass using the median value of blanks (0.31 g for UPAS-collected filters; 0.01 g for PEM-collected filters), and finally dividing by the sampled air volume.

Field equipment (Figure) to be added

4.2.2 Outdoor and indoor (household) air temperature

Hourly outdoor temperature and relative humidity data were obtained from the extensive network of meteorological stations in Beijing (http://beijingair.sinaapp.com). We measured indoor temperature in all participant homes prior to blood pressure measurement. In a random 75% subsample of households in each campaign, we also placed a real-time temperature sensor (iButton DS1921G-F5; Thermochron, Maxim Inc., USA) in the room where participants report spending most of their daytime hours when indoors. Sensors were wall-mounted at a standardized height (~1.5 to 2 meters), away from major heating sources, windows, and doors, and were programmed to log temperature every 125 minutes for up to 4 months to capture the full winter and early spring when heaters may still be intermittently used.

4.2.3 Household stove use using sensors

[...to be completed...]

4.2.4 Questionnaires

Field staff administered household and individual-level questionnaires to assess household demographic information, household assets, house structure, stove and fuel use patterns (including a complete roster of heating methods and their contributions in each room), and smoking. We used Surveybe computer-assisted personal interview (CAPI) software to collect survey data via handheld electronic tablets. Questions were read to participants in Mandarin-Chinese, and their responses were recorded into tablets.

The questionnaire and other study measurements were tested prior to the start of data collection for this study in 12 households located in a Beijing village that was eligible for our study but was instead selected for testing. We used the test village to assess whether the questions were understandable and interpreted as intended and to identify any problems with the study measurements or their implementation. Study protocols were subsequently adapted prior to the start of data collection.

In addition to household and individual participant questionnaires, we also conducted village surveys with one representative from each village committee to inquire about any other policies or programs being implemented in the village (e.g., biomass burning bans) and to understand how the policy was implemented in that village. Committee members answered questions about assignment versus application to the policy, any renovations required by the upper-level government, level of subsidies provided for heating stoves and electricity, and technical and logistic guidance to villagers.

4.2.5 Blood pressure

Following 5 min of quiet rest, at least three brachial and central systolic (bSBP/cSBP) and diastolic (bDBP/cDBP) blood pressures (BPs) were taken by trained staff at 1 min apart on the participant's supported right arm. We used an automated oscillometric device (BP+; Uscom Ltd, New Zealand) that estimates central pressures from the brachial cuff pressure fluctuations. Central pressures were previously validated against invasive cBP measurements (Costello et al. 2015; Lowe et al. 2009). The BP devices were factory calibrated by the manufacturer prior to the start of the first and fourth campaigns. Up to five measurements were taken if the difference between the last two was >5 mmHg or staff were unable to obtain a reading. The BP measurements were conducted in the participant's home and staff were trained to follow strict quality control procedures, including use of an appropriately sized cuff, correct positioning on the arm, both feet on the ground, and ensuring 5 min of quiet rest before measurement (SOP: https://osf.io/gmka5). The average of the final two measurements was used for statistical analysis unless only one BP measurement was obtained (n = 13 observations), in which case, a single measurement was used. The time of day, day of the week, and indoor temperature prior to BP measurement were also recorded.

4.2.6 Self-reported respiratory symptoms and airway inflammation

During questionnaire assessment, participants were asked about recent airway symptoms including cough, phlegm, wheeze, and tightness in the chest using standard American Thoracic Society (ATS) questions that were validated for use in Mandarin-Chinese. In a 25% random subsample of participants in each season, we also measure exhaled nitric oxide, a non-invasive marker of airway inflammation, using a handheld device (NIOX VERO®, Aerocrine, Solna, Sweden), following ATS recommendations and guidelines (ATS/ERS 2005).

4.2.7 Blood inflammatory and oxidative stress markers

Trained nurses collected 4 ml of whole blood in a labeled vacutainer via venipuncture using standard techniques (Tuck 2009). Fasting blood samples were collected in the morning and stored at 4-10°C prior to centrifugation. Two serum aliquots were placed a -30°C freezer for temporary storage.

Collection-to-storage time was <4 hrs for all samples in both campaigns with blood collection. Within 3-5 days of collection, the samples were transported in styrofoam containers with dry ice to a -80°C freezer with a backup generator and alarm system at Peking University. The first aliquot was analyzed for glucose and a complete lipid profile within two months of collection, the results of which were given to participants. The second aliquot was stored in the -80°C freezer for analysis of biomarkers of systemic inflammation (C-reactive protein, interleukin-6, tumour necrosis factor alpha) and oxidative stress (8-hydroxy-2'-deoxyguanosine and malondialdehyde) at the University of the Chinese Academy of Sciences in summer of 2023. These biomarkers were selected because they are associated with the development of cardiovascular disease and events (e.g., Danesh 2008; Pearson 2003; Ridker 2000; 2001; ERF 2012), and both acute and longer-term exposures to air pollution have been associated with changes in inflammatory and oxidative stress markers (e.g., Pope 2004; Rückerl 2007; Rich 2012; Kipen 2010; Huang 2012).

We followed standard methods for analysis (FDA Guidance, 2018). For inflammatory markers (IL-6, TNF- , CRP), the optic densities (OD) of all samples were measured using an automated ELISA reader. Every plate had 8 standard samples used to generate a standard curve that related OD and standard inflammatory marker concentration. A standard curve for each microplate was generated by a computer software program based on a 4-parameter method. Each plate included at least 3 control samples to ensure the stability of standard curves. All samples, standards, and controls were measured in duplicate, and the average was used for statistical analysis. For oxidative stress biomarkers (MDA and 8-OHdG), the chromatographic peak areas of all samples were measured using HPLC with UV detector and HPLC-MS/MS. Every plate had 7 standard samples used to generate a standard curve that related peak area and concentration of each standard oxidative stress marker. A standard curve for each plate was generated using a computer software program based on a linear method. Each plate included at least 3 control samples to ensure the stability of standard curves. Standards and controls were measured in duplicate and samples were measured once due to high precision in a pilot study (Food and Drug Administration 2018).

4.2.8 Anthropometric measurements.

Body weight, height, and waist circumference were measured at the clinic visit in the first two campaigns and in participant homes in the last campaign. Weight was measured in light indoor clothing without shoes in kilograms to one decimal place, using standing scales supported on a steady surface. Height was measured without shoes in centimeters to one decimal place with a stadiometer. Waist circumference was measured at 1 cm above the navel at minimal respiration in centimeters to one decimal place.

4.3 Measuring policy impacts

To understand how Beijing's policy works we used a difference-in-differences (DiD) design (Callaway 2020), leveraging the staggered rollout of the policy across multiple villages to estimate its impact on health outcomes and understand the mechanisms through which it works. Simple comparisons of treated and untreated (i.e., control) villages after the CBHP policy has been implemented are likely to be biased by unmeasured village-level characteristics (e.g., migration, average winter temperature) that are associated with health outcomes. Similarly, comparisons of only treated villages before and after exposure to the program are susceptible to bias by other factors associated with changes in outcomes over time (i.e., secular trends, impacts of the COVID-19 pandemic). By comparing *changes* in outcomes among treated villages to *changes* in outcomes among untreated villages, we can control for any unmeasured time-invariant characteristics of villages as well as any general secular trends affecting all villages that are unrelated to the policy.
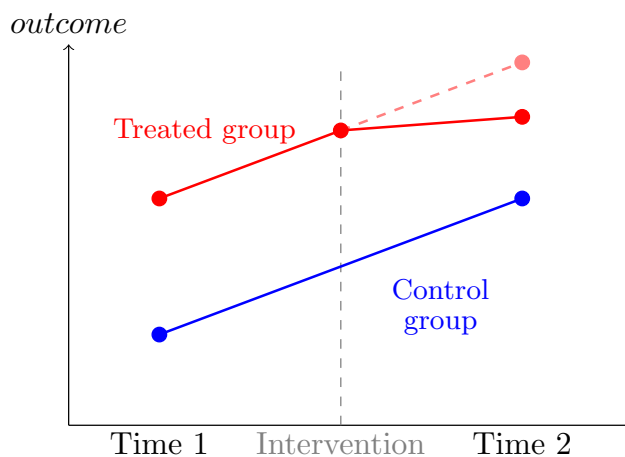

Figure 1: Stylized example of difference-in-differences

The DiD design compares outcomes before and after an intervention in a treated group relative to the same outcomes measured in a control group. The control group trend provides the crucial "counterfactual" estimate of what would have happened in the treated group had it not been treated. By comparing each group to itself, this approach helps to control for both measured and unmeasured fixed differences between the treated and control groups. By measuring changes over time in outcomes in the control group unaffected by the treatment, this approach also controls for any unmeasured factors affecting outcome trends in both treated and control groups. This is important since there are often many potential factors affecting outcome trends that cannot be disentangled from the policy if one only studies the treated group (as in a traditional pre-post design).

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The canonical DiD design (Card and Krueger 1994) compares two groups (treated and control) at two different time periods (pre- and post-intervention, Figure 1). In the first time period both groups are untreated, and in the second time period one group is exposed to the intervention. If we assume that the differences between the groups would have remained constant in the absence of the intervention (parallel trends assumption), then an unbiased estimate of the impact of the intervention in the post period can be calculated by subtracting the pre-post difference in the untreated group from the pre-post difference in the treated group.

However, when multiple groups are treated at different time periods, the most common approach has been to use a two-way fixed effects model to estimate the impact of the intervention which controls for secular trends and differences between districts. However, recent evidence suggests that the traditional two-way fixed effects estimation of the treatment effect may be biased in the context of heterogeneous treatment effects (Callaway and Sant'Anna 2021; Goodman-Bacon 2021)

4.4 Measuring pathways and mechanisms

To estimate how much of the CBHP intervention may work through different mechanisms, we used causal mediation analysis. Causal approaches to mediation attempt to discern between, and clarify the necessary assumptions for identifying, different kinds of mediated effects. Taking as an example the DAG in Figure 2, with T as the policy, M as $PM_{2.5}$, and Y as systolic blood pressure, we can define the controlled direct effect (CDE) as the effect of the CBHP policy on systolic blood pressure if we fix the value of $PM_{2.5}$ to a certain reference level for the entire population. For example, we can estimate the impact of the policy on health outcomes while holding $PM_{2.5}$ at a uniform level of average background exposure, or some other hypothetical level.
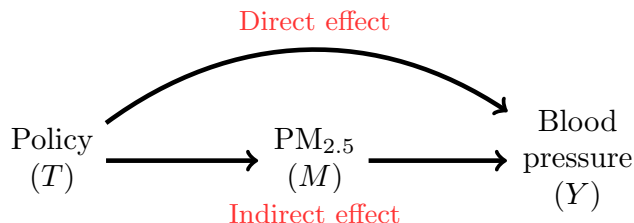
Direct effect

Policy (T) → $PM_{2.5}$ (M) → Blood pressure (Y)

Indirect effect

Figure 2: Example of direct and indirect effects with outcome (Y), treatment (T), and mediator (M)

Although other mediated effects such as "natural" direct and indirect effects are theoretically estimable (VanderWeele 2015), they involve challenging "cross-world" assumptions that are difficult to anchor in policy (Naimi et al. 2014). Other approaches to mechanisms have focused on principal stratification (e.g., Zigler et al. 2016), although conceptual difficulties with identifying the (unverifiable) principal strata make it challenging for questions of mediation. Because controlled direct

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effects are considered more directly policy relevant for public health, we focus on estimating these mediated quantities.

5 Data Analysis

5.1 Total Effect

To estimate the total effect of the policy we used a DiD analysis that accommodates staggered treatment rollout. To allow for heterogeneity in the context of staggered rollout we used 'extended' two-way fixed effects (ETWFE) models (Wooldridge 2021) to estimate the total effect of the CBHP policy. The mean outcome (replaced by a suitable link function $g(\cdot)$ for binary or count outcomes) was defined using a set of linear predictors:

$$Y_{ijt} = g(\mu_{ijt}) = \alpha + \sum_{r=q}^{T} \beta_r d_r + \sum_{s=r}^{T} \gamma_s fs_t + \sum_{r=q}^{T}\sum_{s=r}^{T} \tau_{rt}(d_r \times fs_t) + \varepsilon_{ijt} \tag{1}$$

where Y_{ijt} is the outcome for individual i in village j at time t, d_r represent treatment cohort dummies, i.e., fixed effects for cohorts of villages that were first exposed to the policy at the same time q (e.g., in 2019, 2020, or 2021), fs_t are time fixed effects corresponding to different winter data collection campaigns (2018-19, 2019-20, or 2021-22), and τ_{rt} are the cohort-time *ATTs*. The ETWFE and other approaches that allow for several (potentially heterogenous) treatment effects may also be averaged to provide a weighted *ATT*. Several potential possibilities are feasible, including weighting by treatment cohorts or time since policy adoption (Goin and Riddell 2023)

5.2 Mediation Analysis

As noted above, with respect to the mediation analysis we are chiefly interested in the CDE, which can be derived by adding relevant mediators M to this model. If we also allow for exposure-mediator interaction and potentially allow for adjustment for confounders W of the mediator-outcome effect, we can extend equation Equation 1 as follows:

$$\begin{aligned} Y_{ijt} = g(\mu_{ijt}) = &\alpha + \sum_{r=q}^{T} \beta_r d_r + \sum_{s=r}^{T} \gamma_s fs_t + \sum_{r=q}^{T}\sum_{s=r}^{T} \tau_{rt}(d_r \times fs_t) \\ &+ \delta M_{it} + \sum_{r=q}^{T}\sum_{s=r}^{T} \eta_{rt}(d_r \times fs_t \times M_{it}) + \zeta \mathbf{W} + \varepsilon_{ijt} \end{aligned} \tag{2}$$

where now δ is the conditional effect of the mediator M at the reference level of the treatment (again, represented via the series of group-time interaction terms), and the collection of η terms are coefficients for the product terms allowing for mediator-treatment interaction. Finally, ζ is a vector of coefficients for the set of confounders contained within $\mathbf{W}$.

As noted above, in the staggered DiD framework that allows for heterogeneity we do not have a single treatment effect but a collection of group-time treatment effects that may be averaged in different ways. This extends to the estimation of the CDE, in which case we will also have several CDEs that can be averaged to make inferences about the extent to which the policy's impact is mediated by $PM_{2.5}$. Based on the setup in Equation 2 the CDE is estimated as: $\delta + \eta_{rt} MT$. In the absence of interaction between the exposure and the mediator (i.e., $\eta_{rt} = 0$) the CDE will simply be the estimated treatment effects $\sum_{r=q}^{T} \sum_{s=r}^{T} \tau_{rt}$, i.e., the effect of the policy holding M constant. For a valid estimate of the CDE we must account for confounding of the mediator-outcome effect, represented by W in the equation above. Baseline measures of both the outcome and the proposed mediators inherent in our DiD strategy will help to reduce the potential for unmeasured confounding of the mediator-outcome effect (Keele et al. 2015).
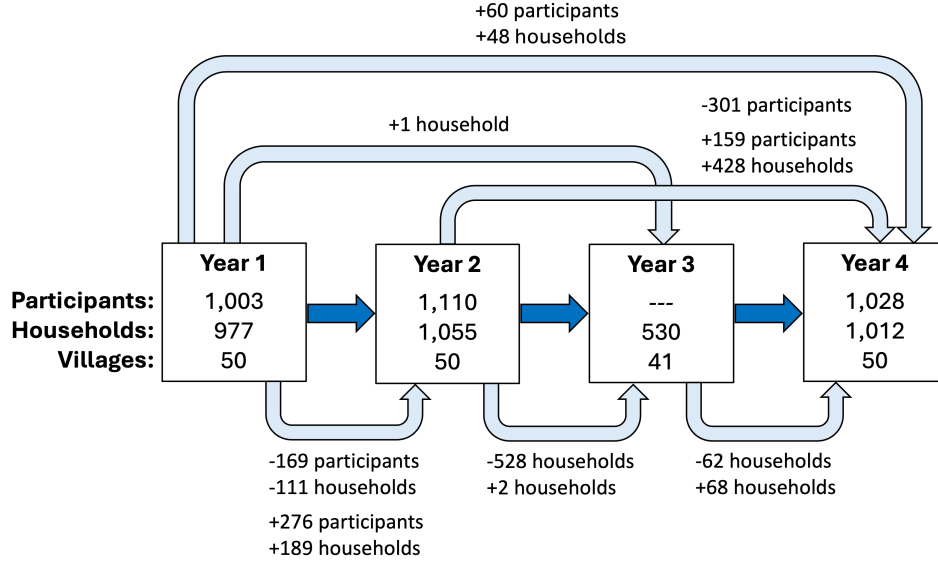
6 Results

Study flowchart

Figure 3: Flow chart of BHET study participation at the participant, household, and village levels across study years. Participation (number of units) in each study year is shown in the white boxes. Additions (+) and losses (-) to the study sample between years are indicated by the light blue arrows. Data collection was limited to household- and village-level environmental measurements due to the COVID-19 pandemic in year 3. We visited 530 households in 41 villages before travel restrictions limited further data collection. This affects the additions and losses to the study sample reported from years 2 to 3 and years 3 to 4.

Figure 3 shows the participation across waves of data collection.

6.1 Description of study sample

Table 2 shows the distribution of selected demographic, health, and environmental characteristics from the baseline survey, prior to any villages being enrolled in the ban. We provide means and standard deviations separately for villages that eventually enter into the ban with those that never do so. As noted above, although our DiD identification strategy allows for fixed differences between treated and untreated villages, overall the differences at baseline are generally small and the groups seem well balanced on most measures, with the exception of personal $PM_{2.5}$ exposure, which was lower in villages that were eventually treated.

Table 2: Descriptive statistics for selected demographic, health, and environmental measures at baseline, by treatment status

	Never treated (N=603)		Ever treated (N=400)			
	Mean	Std. Dev.	Mean	Std. Dev.	Diff. in Means	Std. Error
Demographics:						
Age (years)	59.9	9.4	60.4	9.2	0.5	0.6
Female (%)	59.5	49.1	59.1	49.2	-0.4	3.2
No education (%)	11.5	31.9	12.3	32.9	0.9	2.1
Primary education (%)	75.5	43.0	77.6	41.7	2.1	2.8
Secondary+ education (%)	12.6	33.2	9.8	29.7	-2.9	2.0
Health measures:						
Never smoker (%)	61.9	48.6	59.5	49.1	-2.4	3.2
Former smoker (%)	11.9	32.4	15.1	35.8	3.2	2.2
Current smoker (%)	26.2	44.0	25.4	43.6	-0.8	2.8
Never drinker (%)	55.9	49.7	52.5	50.0	-3.4	3.2
Occasional drinker (%)	26.0	43.9	25.5	43.6	-0.5	2.8
Daily drinker (%)	17.8	38.3	21.9	41.4	4.1	2.6
Systolic (mmHg)	131.4	16.8	128.7	14.3	-2.7	1.0
Diastolic (mmHg)	82.7	11.6	82.1	11.3	-0.6	0.8
Waist circumference (cm)	87.7	10.5	85.4	9.5	-2.3	0.8
Body mass index (kg/m2)	26.3	3.7	25.8	3.6	-0.5	0.3
Frequency of coughing (%)	18.7	39.0	19.7	39.8	1.0	2.6
Frequency of wheezing (%)	6.2	24.2	6.6	24.8	0.3	1.6
Shortness of breath (%)	29.2	45.5	34.3	47.5	5.1	3.0
Chest trouble (%)	11.6	32.0	14.1	34.9	2.5	2.2
Any respiratory problem (%)	50.6	50.0	54.3	49.9	3.7	3.2
Environmental measures:						
Temperature (°C)	13.8	3.6	13.5	3.3	-0.3	0.2
Personal PM2.5 (ug/m3)	150.2	300.3	103.8	107.3	-46.3	19.1

Includes all individuals sampled at each of 3 waves.

6.2 Summary of PM and BC measurements

At baseline, fine particulate matter ($PM_{2.5}$) and black carbon (BC) concentrations were highest, on average, for personal measurements compared to indoor and outdoor measurements, with indoor levels being higher than outdoor levels (Figure 4). This trend (personal > indoor > outdoor) was observed among households in treated and control villages. Personal, indoor, and outdoor geometric mean (95% confidence interval) concentrations of $PM_{2.5}$ were 72 (65,80), 45 (39,53), and 31 (28,35), respectively, and elevated relative to health-based guidelines. The current World Health Organization (WHO) guidelines state that annual average concentrations of PM2.5 should not exceed 5 µg/m3, while 24-hour average exposures should not exceed 15 µg/m3 more than 3 - 4 days per year. Interim targets have been set to support the planning of incremental milestones toward cleaner air, particularly for cities, regions and countries that are struggling with high air pollution levels. For PM2.5 these are: 35 µg/m3 annual mean, 75 µg/m3 24-hour mean; 25 µg/m3 annual mean, 50 µg/m3 24-hour mean; 15 µg/m3 annual mean, 37.5 µg/m3 24-hour mean; 10 µg/m3 annual mean, 25 µg/m3 24-hour mean.

		Overall	Treated	Control
Personal	$PM_{2.5}$	72 [65, 80]	64 [54, 74]	79 [69, 89]
	BC	2.6 [2.4, 2.8]	2.1 [1.8, 2.5]	2.9 [2.7, 3.3]
Indoor*	$PM_{2.5}$	45 [39, 53]	43 [33, 56]	47 [38, 58]
	BC	1.6 [1.3, 2.0]	1.4 [1.0, 1.9]	1.8 [1.4, 2.3]
Outdoor	$PM_{2.5}$	31 [28, 35]	24 [19, 31]	36 [32, 41]
	BC	1.3 [1.1, 1.4]	1.2 [1.0, 1.4]	1.3 [1.2, 1.6]

*Indoor shows data collected in S2 because we did not measure indoor air pollution in S1.

Figure 4: Geometric mean and 95% Confidence Intervals for filter-based air pollutant concentrations at baseline.

6.3 Policy uptake

Each year of the study, participants reported what energy types they used for space heating in winter. Based on these data, heating energy types were classified into four categories: exclusive use of a heat pump ('heat pump exclusively'), use of a heat pump and a kang ('heat pump with kang'), use of solid fuel with use of electric heating devices that were not heat pumps ('solid fuel with electricity (not heat pump)'), and exclusive use of solid fuel. In villages treated under the policy, Figure 5 shows meaningful transitions from solid fuel to heat pumps was observed. For example, in villages treated in S2, over 90% of households used heat pumps in S2, increasing to 96% in S4, while only 3% used heat pumps in S1. Conversely, the use of solid fuel decreased from

97% in S1 to 8% in S2 and 3% in S4. Villages treated in S4 initially relied heavily on solid fuel, with approximately 90% of households reporting use of solid fuels in S1 and S2. However, after treatment, 94% of households in these same villages reported using heat pumps in S4.

In untreated villages, an active transition from solid fuel to clean energy was also observed. The use of heat pumps increased gradually from 5% in S1 to 10% in S2 and 25% in S4. The percentage of households using solid fuel with electric devices remained relatively stable, ranging from 64% to 70%. However, the exclusive use of solid fuel decreased from 30% in S1 to 7% in S4. We also observed a substantial decline in self-reported coal use when villages entered into the CBHP policy (Appendix Figure A1)
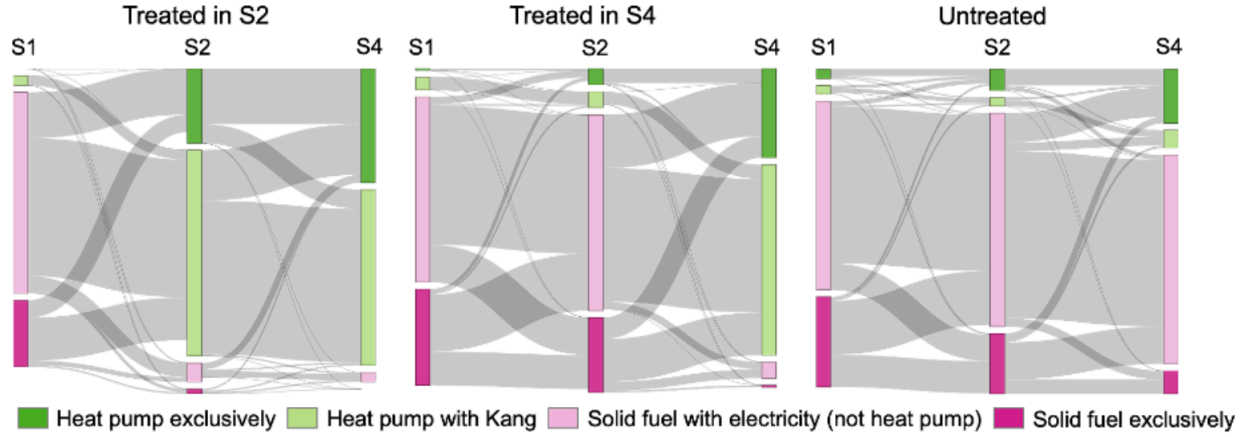

Figure 5: Transitions to different energy sources across study seasons

6.4 Aim 1: Policy impacts and potential mediation

6.4.1 Impact of policy on PM mass

In estimating the treatment effect on indoor and outdoor air pollution, we evaluated both 24-h mean values (specifically, the same 24-h period when personal exposure samples were collected in each village) and seasonal mean values (with 'season' defined from Jan. 15th to Mar. 15th) of PM2.5 data collected in each village. For estimating the treatment effect on personal exposure to PM2.5 and black carbon (BC), the results from the filter-based measurements that were collected for a 24-h period were used for analysis. We estimated the basic ETWFE models, as well as ETWFE models further adjusted for covariates, including temperature, relative humidity, wind speed, boundary layer height, wind direction, and the mean quantity of wood burned in each village).
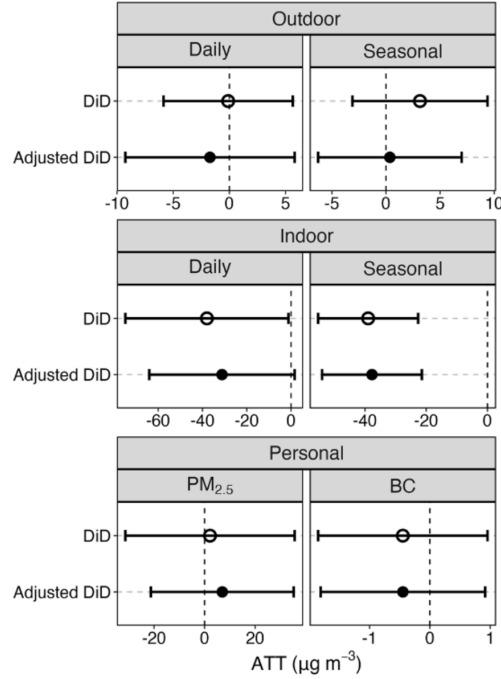
Figure 6: Treatment effect on outdoor and indoor PM2.5, as well as personal exposure to PM2.5 and black carbon. Outdoor and indoor PM2.5 were derived from sensor measurements after being adjusted based on co-located gravimetric PM2.5 measurements. 'Daily' indicates the mean PM2.5 concentrations during the 24 hours when personal exposure samples were collected in each village. 'Seasonal' indicates the seasonal mean PM2.5 concentrations in each village, from Jan. 15th to Mar. 15th. 'Did' represents the DiD analysis without any covariates, while 'Adjusted DiD' represents DiD analysis with covariates.

Treatment was associated with reductions in both seasonal and 24-h indoor PM2.5 means (Figure 6). On average, treatment was associated with a decrease in 24-h indoor PM2.5 of 38 [75, 1] g/m3. After adjusting for covariates such as outdoor temperature, dewpoint, household smoking status, and the number of residents in each household, the treatment effect decreased to 31 [64, -2] g/m3. The treatment effect on seasonal indoor PM2.5 (39 [55, 23] g/m3) remained consistent even after covariate adjustment. This finding likely reflects the direct benefit of the policy in replacing coal stoves, thereby improving indoor air quality.

Overall we found little evidence of an impact of the CBHP policy on 24-h and seasonal outdoor (local community-level) $PM_{2.5}$, as well as limited evidence of any impact on personal PM2.5 and BC. Treatment was associated with lower, but statistically imprecise, personal 24-h BC exposures. This finding would be consistent with the expectation that the policy contributed to reducing air

pollutant emissions from solid fuel burning, as BC serves as a potential indicator of such combustion, particularly in our study settings.

6.5 Impact of policy on health outcomes

	DiD ATT (95% CI), mmHg	Adjusted DiD* ATT (95% CI), mmHg
Systolic BP		
Brachial	-0.79 (-2.63, 1.04)	-1.4 (-3.31, 0.51)
Central	-1.04 (-2.82, 0.73)	-1.56 (-3.4, 0.28)
Diastolic BP		
Brachial	-1.29 (-2.62, 0.04)	-1.6 (-2.96, -0.25)
Central	-1.35 (-2.66, -0.04)	-1.66 (-2.97, -0.34)
Pulse pressure		
Brachial	0.5 (-0.71, 1.7)	0.21 (-1, 1.41)
Central	0.31 (-0.85, 1.46)	0.1 (-1.01, 1.2)
BP Amplification		
Pulse pressure amplification	0.001 (-0.012, 0.014)	0.000 (-0.012, 0.012)
Systolic BP amplification	0.002 (-0.002, 0.005)	0.001 (-0.002, 0.004)

Figure 7: Overall impacts of the 'coal-to-clean energy' policy on BP, pulse pressure, and BP amplification

Figure 7 shows the impacts of the policy in basic ETWFE models and models further adjusted for age, sex, waist circumference, smoking, alcohol consumption, and use of blood pressure medication. Overall exposure to the CBHP policy demonstrated reductions in blood pressure of approximately 1.5mmHg on systolic and diastolic BP, but we found little evidence of a meaningful impact on pulse pressure or BP amplification.

Table 3 shows the impacts on respiratory outcomes. In both basic and covariate-adjusted ETWFE models we found that exposure to the CBHP policy reduced self-reports of any poor respiratory symptoms by around 7 percentage points. This was largely through reductions in reports of having chest trouble or difficulty breathing several or most days of the week.

Table 3: Marginal effect of policy on self-reported respiratory symptoms from basic and adjusted DiD models

	Basic DiD			Adjusted DiD		
Frequency of:	Estimate	LL	UL	Estimate	LL	UL
Any respiratory symptom	-0.07	-0.14	-0.01	-0.08	-0.15	-0.01
Coughing	-0.02	-0.06	0.03	-0.02	-0.07	0.03
Phlegm	-0.01	-0.06	0.03	-0.02	-0.06	0.03
Wheezing attacks	0.00	-0.04	0.04	0.00	-0.04	0.04
Trouble breathing	-0.05	-0.12	0.02	-0.05	-0.12	0.02
Chest trouble	-0.07	-0.13	-0.01	-0.06	-0.12	-0.01

6.6 Mediated impact on blood pressure

As noted above, we aimed to assess whether any health impacts of the CBHP policy may work specifically through pathways involving changes in $PM_{2.5}$ and indoor temperature. Below we show in results from several mediation models. We evaluated potential mediation for each mediator separately and in a single model accounting for multiple mediators, and we set the values of both mediators to the WHO mean annual interim $PM_{2.5}$ and indoor temperature guidelines. Given that we did not find evidence of any policy impact on BP outcomes other than systolic and diastolic BP, we did not undertake mediation analysis for other outcomes. In Table 4 we show that conditioning on indoor PM and indoor temperature largely explains the entire total effect of the CBHP policy on blood pressure for systolic BP, and roughly half of the total effect for diastolic BP.

Table 4: Controlled direct effects for the CBHP policy

		CDE Mediated By:		
Outcome	Adjusted Total Effect	Indoor PM	Indoor Temp	PM + Temp
Brachial SBP	-1.4 (-3.31, 0.51)	-0.93 (-3.05, 1.2)	-0.43 (-2.35, 1.49)	0.13 (-2.02, 2.28)
Central SBP	-1.56 (-3.4, 0.28)	-1.02 (-3.13, 1.08)	-0.54 (-2.39, 1.3)	0.07 (-2.05, 2.19)
Brachial DBP	-1.6 (-2.96, -0.25)	-1.3 (-2.81, 0.21)	-1.02 (-2.45, 0.42)	-0.65 (-2.25, 0.94)
Central DBP	-1.66 (-2.97, -0.34)	-1.31 (-2.81, 0.19)	-1.08 (-2.48, 0.32)	-0.68 (-2.27, 0.9)

6.7 Aim 2: Source contributions

The model diagnostics for the 3 to 6-factor PMF solutions are given in Figure 8. Model fit was assessed using Q/Qexp (how our model fit divided by the expected fit). As the change in Q/Qexp

decreases as more factors are added, the model may be fitting additional sources that do not improve the overall fit. The largest change in Q/Qexp was from 3 to 4 sources (6.24 to 5.37) while the changes moving from 4 to 5 and 5 to 6 are similar which suggests that 4 factors is sufficient to explain the variation in our data. We assessed the random error in our model by randomly sampling blocks of data, fitting new models with the blocks, and comparing how the source profiles compared to that of the original model (BS mapping). The 3 and 4 factor solutions had high BS mapping (all factors found in $> 96.5\%$ of bootstrap runs). The additional sources identified in the 5 (lead) and 6 (chloride) factor solutions have low bootstrap mapping ($> 72\%$) which means those solutions are not as consistent as the 3 and 4 factor solutions. The possibility that multiple, different, solutions could result in the same Q value was assessed using displacement. The displacement approach takes the original factor profiles and modifies the values for each species up or down to maintain a small change in Q, reruns the solution with the new species values, then compares the profiles of the new model to the original. Any swaps indicate that small changes in the species values could result in factor profiles that look different from the original solution, and that the original solution is unstable. None of the factors in any of the solutions discussed were swapped during displacement which indicates that all of the potential solutions are stable. Based on the Q/Qexp, BS mapping, and interpretability of the factors, the 4 factor solution was chosen for this dataset.

Diagnostic	3	4	5	6
Qexp	27936	26052	24168	22284
Qtrue	187681	147796	123236	100316
Qrobust	174407	139910	117082	95932.5
Qr/Qexp	6.24	5.37	4.84	4.30
Q/Qexp > 6	wi-Ca, ns-S, ws-Na, ws-Ca, Al, Cl, Pb	ns-S, Na, Al, Cl, Pb, Nitrate	Nitrate, ws-Na, Al, Chloride	Nitrate, ws-Na, Al
DISP % dQ	<0.1%	<0.1%	<0.1%	<0.1%
DISP swaps	0	0	0	0
BS mapping < 100%	Dust- 98.5%	Transported dust- 95%, Dust- 96.5% Sulfur secondary- 97.5% Mixed combustion- 96.5%	Transported dust- 86% Mixed combustion- 87% Dust- 86% Lead- 55%	Transported dust- 84% Mixed combustion- 87.5% Dust- 81.5% Lead- 72% Chloride- 61.5% Sulfur secondary- 98.5%

Figure 8: PMF error estimation diagnostics.

The source profiles for the four-factor solution are presented in Figure 9. The first source was identified as dust by high percentages of crustal elements like wi-Ca, Si, and wi-Mg. The second source was constituted of non-sulfate sulfur as well as secondary inorganic ions (ammonium, nitrate,

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and sulfate). Non-sulfate sulfur is a tracer for primary coal combustion, while secondary inorganic ions indicate a secondary source. Since coal combustion is a major source of energy in our study area, it is likely that the second source is a mixture of primary and secondary emissions that originate from coal and other sulfurous fuel combustion. Additionally, the mean source contribution of the second source is higher in outdoor than personal exposure measurements. Secondary formation occurs outdoors in the presence of sunlight, so higher outdoor concentrations compared to personal exposure further support our naming the second source and sulfur secondary. The third source had high percentages of ws-Ca nd Al, which in our study region, has been found to be indicative of transported dust from dust storms that can occur in the spring. While our samples were collected during winter months only, it is possible that transported dust from previous years still remained. The fourth source was characterized by high percentages of tracers for both coal (OC, wi-K, chloride, Pb) and biomass combustion (EC, ws-K). Coal and biomass combustion is common in our study setting so this source is likely a mixture of the two combustion sources.
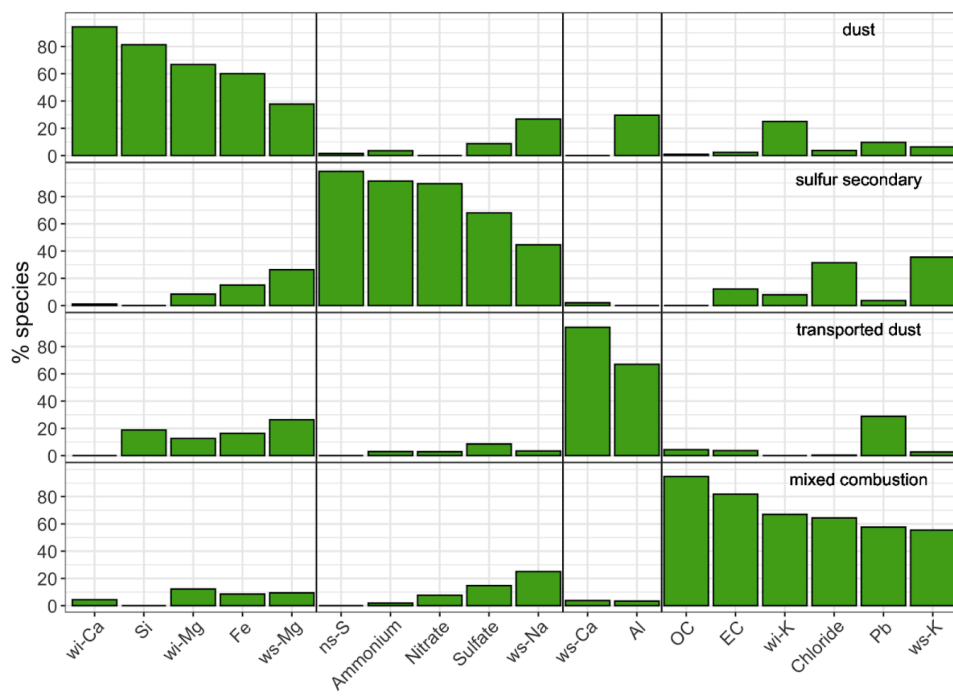

Figure 9: Source profiles for the 4-factor PMF solution to the sum of elements, ions, elemental carbon, and organic carbon for outdoor and personal PM2.5 exposure measurements. The lines separate the major contributing species to each source

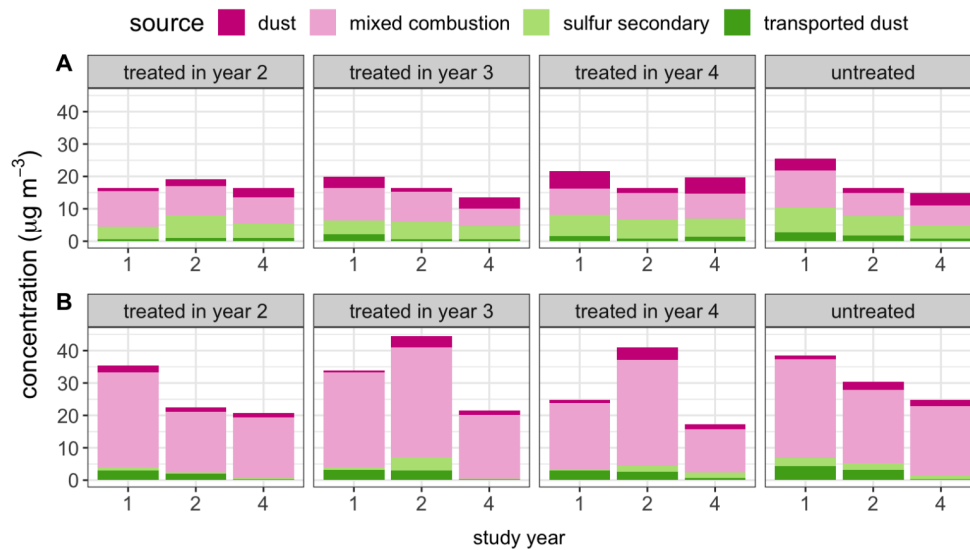We extend the source profiles across the different treatment cohorts in Figure 10.

Figure 10: Arithmetic mean dispersion normalized source contributions found from the 4-factor PMF solution for A outdoor and B personal PM2.5 exposure samples by year the group received treatment.

6.8 Aim 3

- Table of mediated health effects by source contribution (coal and biomass)

7 Discussion and Conclusions

- Generally describing high take-up of the policy
- Reductions in indoor $PM_{2.5}$ but not personal or outdoor
- Reduction in blood pressure and self-reported respiratory symptoms

Other relevant results (Tables or figures in SI)

Policy impacts on other relevant outcomes:

- Temperature
- Heating room
- Well-being

8 Implications of Findings

9 Data Availability Statement

- Description of datasets and code available on our project page at the Open Science Foundation

10 Acknowledgements

To come...

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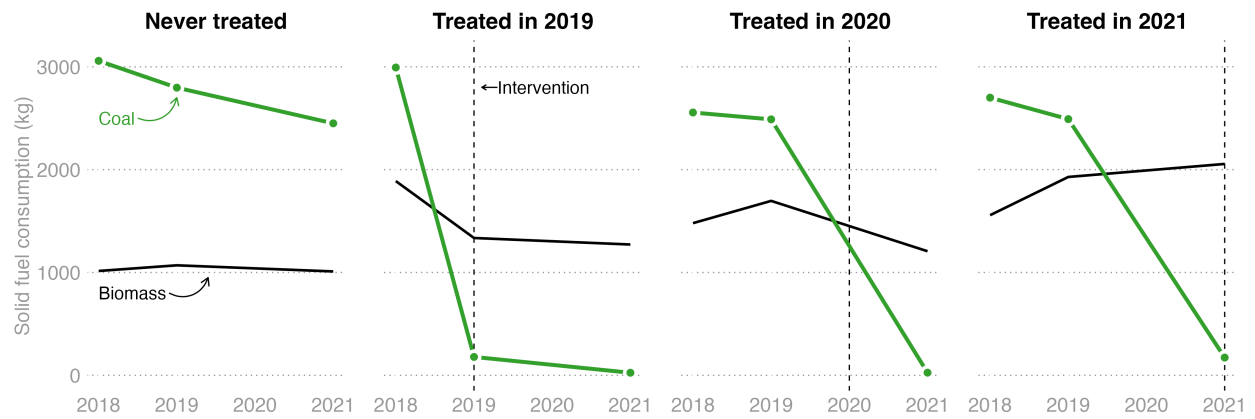
Appendices

Figure A1: Trends in self-reported coal and biomass, by treatment season

About the authors

Other publications