2019-02

Working paper. Economics

ISSN 2340-5031

COOKING THAT KILLS: CLEANER ENERGY, INDOOR AIR POLLUTION, AND HEALTH

Imelda

uc3m | Universidad Carlos III de Madrid

COOKING THAT KILLS: CLEANER ENERGY, INDOOR AIR POLLUTION, AND HEALTH[*]

Imelda[†]

January 14, 2019

Abstract

Cooking with dirty fuel is known to be one of the biggest sources of indoor air pollution in developing countries. I estimate the health impact of indoor air pollution using a nationwide fuel-switching program, the largest household energy transition project ever attempted in the developing world, affecting more than 50 million homes in Indonesia. This program focused on replacing a dirty cooking fuel, kerosene, with cleaner cooking fuel, liquid petroleum gas (LPG). I use a difference-in-differences design with time-varying program intensities to capture the dynamic increase in the households' access to LPG. A 10-percentage-point increase in the program intensity – measured by the number of free initial LPG packages distributed – reduces infant mortality rate by 3.3 percentage points, or 1.2 infants per 1,000 live births annually. This study highlights the fact that adopting cleaner energy can have a substantial health impact beyond what is currently known.

JEL classification: I12, J13, O15, Q48, Q52
Keywords: indoor air pollution, infant mortality, kerosene, LPG, Indonesia

[*] For their comments and suggestions, I thank Inessa Love, Michael J. Roberts, Timothy Halliday, John Lynham, Natalia Fabra, Budy P. Resosudarmo, Blane Lewis, Arya Gaduh, Irma Clots Figueras, Jaime Millan, Teresa Molina, Wayne Liou, and seminar participants at Universidad Carlos III de Madrid, UNU-WIDER, Camp Resources NC State, Australian National University, Australian Development Economics Workshop, American Agriculture and Applied Economics Meeting, CSWEP "Health and Development" 2018 AEA Session. For their help with data, I thank R. Zulfikar and staff members at Pertamina.

[†] Universidad Carlos III de Madrid, Calle Madrid, 126, 28903 Getafe, Spain. Email: iimelda@eco.uc3m.es.

I. Introduction

To what extent does indoor air quality affect health? While most of the existing studies focus on the impact of outdoor air pollution on health, people tend to be more exposed to indoor than outdoor pollution because individuals spend more time indoors and closer to the source of indoor pollutants (Bennett et al., 2002; Oliveira et al., 2017; Requia et al., 2017). Indeed, millions of lives are lost annually due to illnesses attributable to indoor air pollution (IHME, 2016).

Despite the high economic cost associated with indoor air pollution, its causal impact on health is severely understudied, mainly due to data and methodological challenges (Duflo et al., 2008). The health effects from indoor air pollutants are often confounded by the unobserved determinants of health that are associated with its exposure. Moreover, as current studies largely focus on adult diseases, the health effects can be biased by individuals' exposure to the pollutants that occurred many years ago. Plus, large variations in indoor air pollution exposure across homes and individuals, coupled with the smaller number of observations, lead to imprecise estimates in the existing studies.

In this study, I estimate the impact of indoor air pollution on health using a nationwide fuel-switching program, from a relatively dirty cooking fuel (i.e., kerosene) to a much cleaner cooking fuel (i.e., LPG) in Indonesia.[1] This program reached more than 70% of the total population in Indonesia, resulting in a sharp drop in kerosene consumption and an increase in LPG consumption, contemporaneously. I use the program as a proxy for the changes in individual exposure to indoor air pollution.[2] By exploiting exogenous variations in the program intensity, measured by the free initial LPG packages distributed, I am able to show the causal effect of indoor air pollution on infant mortality, thus overcoming methodological challenges of earlier studies.

The study of infant mortality is an important contribution from this paper. Focusing on infants helps establish a strong and immediate connection between pollution exposure and health as it excludes the unknown accumulation of pollution exposure over one's lifetime (Chay and Greenstone, 2003; Currie and Neidell, 2005; Currie and Walker, 2011), one of the main sources of bias of earlier studies. Current

[1] Cooking with dirty fuels is often claimed to be one of the biggest sources of indoor air pollution in developing countries (Martin et al., 2011). For example, $PM_{2.5}$ exposure from stoves that use kerosene, one of the dirty fuels, is similar to 24-hour $PM_{2.5}$ concentrations in Scottish homes that smoke ten cigarettes per day (Semple et al., 2012).

[2] Most exposure studies have reached a common conclusion that kerosene-using stoves emit more pollutants compared to LPG-using stoves (see Bryden et al. 2015 for a review), suggesting that this program that aimed to replace kerosene with LPG reduces indoor air pollution.

attempts to study infant mortality are hindered by a limited number of observations paired with short study periods.[3] A nationally scaled policy experiment that has much more observations than previous studies allows me to provide novel empirical evidence on infant mortality. Moreover, infants spend most of their time indoors and are particularly more vulnerable to environmental risk. They are also more likely to be disproportionately affected by the indoor air pollutants starting at the fetal stage because pregnant mothers spend much more time indoors compared to the other household members.

This policy experiment that affected more than 50 million homes also allows me to mitigate the other main challenge: measurement error about the individual exposure to indoor pollution, which often leads to a severe attenuation bias (Sheppard et al., 2012). Especially in an indoor air pollution context, the attenuation bias created by measurement errors can be exacerbated by the large variation in indoor pollution exposures.[4]

Indonesia provides a compelling setting to study indoor air pollution that is associated with a cleaner energy transition. Foremost, it is the fourth most populated country in the world, with 3.5% of the global population. However, only 10% had access to clean cooking fuels in 2005. Additionally, in 2007, pressured by a rising cost of subsidizing kerosene, the Vice President of Indonesia set an ambitious goal of converting 42 million households to using LPG by 2012 (WLPGA, 2012). As a result, the program implementation was undertaken as a learning-by-doing basis due to time and logistical constraints. This gives rise to plausibly exogenous variation in the amount of initial LPG distributed.[5] Finally, the LPG adoption rate was very high due to strict enforcement through kerosene supply reduction. Therefore, using this program enables me to improve previous experimental studies that face noncompliance issues (Hanna et al., 2016).

I compare changes in infant mortality in treatment districts to changes in infant mortality in control districts, using three rounds of Indonesian Demographic and Health Surveys. The main threat to causal

[3] Infant mortality studies need much more observations over a long period of time. However, the operational costs of measuring indoor air exposures are high. Hence, many studies have limited sample sizes and short study periods (i.e., up to two years). Only Hanna et al. (2016) investigate the long-term health effects of improved cook-stoves over four years.

[4] This attenuation bias increases in the fraction of total variations observed (Greenstone and Gayer, 2009). The large variation is mainly due to the fact that indoor pollution exposures measured in one home can be very different from its neighbors, unlike outdoor pollution exposures. One monitoring station measures outdoor exposure to hundreds of homes, while one monitoring device in one home measures exposure to one home.

[5] First, this program commenced after only 8 months of a simple feasibility study and one-month of market trial. Furthermore, in the first two years after the program started, there was a strong resistance from the community (i.e., mass protests and negative public opinion in the media) and simultaneous kerosene and LPG scarcity. Only after about two years, the program no longer had any major operational issues (Budya and Arofat, 2011).

identification is that the timing of the program implementation might have been associated with unobserved factors that otherwise influenced infant mortality in the treatment districts. I show, however, that similar pre-implementation trends in infant mortality exist in both treatment and control districts. I also show that the program timing has no association with trends in the observable characteristics at baseline, suggesting that the treatment districts are a valid counterfactual for the control districts in the absence of the program. Further, I compare changes in infant mortality within districts with varying degrees of program intensities. Lastly, the results are largely robust to a wide range of alternative specifications that control for omitted variables and unobserved characteristics of mothers that could confound the estimates.

I find that a 10-percentage-point increase in the free initial LPG distributed reduces infant mortality rate by 3.3 percentage points, or 1.2 infants per 1,000 live births annually. This reduction in the infant death mostly occurred within the first day of birth, suggesting that fetal exposure to kerosene-related pollutants appears to be an important channel. Compared to the literature on ambient air pollution, the effects observed in this study are smaller, which can be explained by the modest indoor air quality improvement from the kerosene to LPG transition (i.e., LPG is only marginally superior to kerosene). In contrast, the program has no effect among kerosene users or households without access to electricity that still rely on kerosene for lighting purposes. These results serve as placebo checks, mitigating concerns that the program intensity is confounded with unobservable determinants of infant mortality.

I next investigate several channels for the impact of the program on health and find that the improvement in indoor air quality is the most relevant channel. First, switching from a kerosene stove to an LPG stove is associated with a 30% reduction in the fine particulates ($PM_{2.5}$) personal exposure (Andresen et al., 2005) and 20% lower carbon monoxide (CO) concentration (Smith et al., 1999).[6] Second, I discuss changes in expenditures, fuel stacking, and avoidance behavior as potential alternative mechanisms. However, none of these seem to have played a major role.

This paper contributes to the growing literature on the causal link between pollution and health (see Greenstone and Jack 2015 for a summary of this research). Existing studies on indoor air pollution have adopted an instrumental variable approach to address the endogeneity of indoor pollutant exposures.

[6] Due to lack of indoor air quality measurement in Indonesia, I refer $PM_{2.5}$ and CO exposures from LPG relative to kerosene from other exposure studies.

For example, Pitt et al. used gender-specific hierarchies as an instrument for pollution exposure to study its health impact in Bangladesh.[7] I use a unique policy experiment to address the unobserved time-varying factors that determine infant mortality. My estimates include potential indoor air pollutants that escape outdoors and contribute to local outdoor air pollution. By using the program intensity, I also improved on previous work which used a simple binary treatment, which is common in the relevant literature (Imelda, 2018a).

My findings shed light on an important, ongoing policy debate about the optimal policies for addressing indoor air pollution (i.e., to rely on advanced technology to make available dirty fuels emit less pollution or to provide access to clean fuels).[8] In a real-world setting, behavioral aspects[9] may obscure potential impacts. Nonetheless, I show that when a cleaner fuel is provided and people are adopting it, this can lead to significant health impacts.

The rest of the paper is organized as follows: Section II provides some background about the program; Section III describes the data and the empirical strategy; Section IV shows my main results; Section V discusses the mechanism; Section VI shows the robustness checks and the heterogeneous effects; and Section VII concludes.

II. Indonesia's Cooking Fuel Conversion Program

Kerosene is a petroleum product that has a strong odor similar to gasoline. It is one of the most important household commodities used for cooking and lighting in Indonesia since the 1960s. A decline in crude oil production and rising demand for domestic fuel consumption have shifted Indonesia from major oil exporter to a net oil importer. After oil prices increased in 2006, subsidizing kerosene had become a major burden to the Indonesian government (Budya and Arofat, 2011). Hence, in 2007, the government introduced the Kerosene to LPG conversion program.

LPG is a mixture of propane and butane. It is known as a cleaner-burning and more efficient fuel compared to kerosene because of the higher combustion efficiency (Kimemia and Van Niekerk, 2017). It produces less CO and less $PM_{2.5}$, relative to kerosene (Smith et al., 1999; Andresen et al., 2005;

[7] In another example, Silwal and McKay (2015) used distance from the community to the nearest market as an instrument for indoor air pollution from using firewood for cooking.

[8] See Smith and Sagar (2014) for a review.

[9] For example, low willingness to pay for clean fuels (Mobarak et al., 2012) and a lack of understanding on the proper use of the clean technology (Hanna et al., 2016) can hinder households' adoption of it.

Bryden et al., 2015). The Indonesian government chose LPG as the fuel to replace kerosene mainly because of its price and its existing infrastructure (Thoday, 2018).

Indonesian government encouraged households to switch their cooking fuel to LPG by (1) distributing one free initial LPG package to each eligible household, (2) increasing the availability of LPG refills under a subsidized price, and (3) reducing the supply of kerosene gradually. Figure 1 shows the total population based on households' primary cooking fuel reported in the census, during 1971-2010. The share of households who used kerosene was stable prior to the program. However, after the program rolled out in 2007, the total population who use kerosene dropped from 42% to 12%, and the total population who used LPG increased from 9% to 46% within only three years.

The main purpose of the program is to reduce kerosene subsidy. The Ministry of Energy and Mineral Resources, the program coordinator, mainly targeted districts that have a high level of kerosene use and are near LPG infrastructure. Therefore, it is not surprising that big cities with large population such as Jakarta and Denpasar are among the early targeted districts, while the remaining districts that are less densely populated and farther from the ports are among the late targeted districts (see Figure 2). Local governments had a key role in determining households eligibility.[10] These eligible households received one initial free LPG package (one LPG canister, a single LPG stove, hose, and regulator) and were allowed to refill the LPG canister under the subsidized price.[11]

The reduction in the kerosene consumption and the increasing use of LPG occurred contemporaneously, as shown in Figure 3 and 4 highlight the rapid expansion of the program across districts within a relatively short time. Figure 3 shows the changes in the district's LPG and kerosene share from 2005 to 2010.[12] In contrast, the share of biomass users is unaffected. The cost of using biomass for cooking is very low, often free, thus households that mainly use biomass for cooking do not have strong incentives to switch to LPG.[13] Similarly, Figure 4 shows a rapid expansion of the program, reflected by a contemporaneous increase and decrease of LPG and kerosene consumption from 2006 to 2012.

[10] For example, in Jakarta, households that have not used LPG stove before and have an income of less than USD 167 per month are eligible for the program (Thoday, 2018).

[11] Only the 3 kg-sized LPG cylinder is allowed to be refilled under the subsidized price. Other LPG sizes, circulated before the program, are not eligible to be refilled under subsidized price. For more details on the program, I refer readers to Budya and Arofat (2011) and Thoday (2018).

[12] The changes in cooking fuel t in district r and year t is calculated as

$$\frac{\sum \text{Population}_{f,r,t=2010}}{\text{Population}_{r,t=2010}} - \frac{\sum \text{Population}_{f,r,t=2005}}{\text{Population}_{r,t=2005}}, \text{ where } f \text{ is either LPG or kerosene.}$$

[13] Most wood fuel users get the wood from their own gardens (Andadari et al., 2014).

III. Data and Empirical Strategy

III.A Program Variables

Data on the program implementation comes from Pertamina, Indonesia's national oil company appointed as the sole LPG supplier for the program. With this data, I construct two treatment variables: (1) a binary treatment variable indicating if district r has any LPG packages distributed in the year t (later called program dummy) and (2) per capita program intensity (later called program intensity). The program intensity is calculated as the number of initial free LPG packages distributed at year $t-1$ in the district r divided by total working age population[14] in district r in 2010, expressed as follows.

$$\text{Program intensity}_{rt} = \frac{\text{LPG packages}_{rt-1}}{\text{Population (15-64)}_{r,t=2010}} \tag{1}$$

Figure 5 shows program intensity over time for each district, calculated using Equation 1. As shown in the figure, the number of LPG packages are distributed to more districts after 2008. The largest expansion occurred in 2010, which covered more than 150 districts out of 511. The correlation coefficient between program dummy and program intensity is 0.58. However, the program intensity provides a precise diffusion of districts access to LPG than the program dummy does.[15]

It is important to highlight several other concurrent changes or policies that are suspected to be associated with the timing of the program and the program intensity. First, in 2007, the Indonesian government implemented two conditional cash transfer programs (CCT): Program Keluarga Harapan (PKH) and PNPM Generasi (Sparrow et al., 2008; Alatas et al., 2011). Even though these programs were targeted on improving health, they used randomization and geographic targeting.[16] Moreover, the program has neither an impact on infant mortality nor on children's nutritional status (Alatas et al., 2011). The program increased health care utilization, but not the quality of the health care (Triyana, 2016). Second, the program might lead to an expansion in the LPG infrastructure. However, to fulfill

[14]The working age group, people within 15-64 years old, is the group who are likely exposed by the program. They are more likely to have influence in the household decisions, compared to the remaining population (i.e., younger than 15 years old and older than 64 years old). The population census in 2010 is chosen as the denominator since it has a more complete data on districts population compared to population census in 2005. The total population in 2005 and 2010 are 226,712,730 and 242,524,123, respectively.

[15]There are several other ways to measure program intensity (e.g., log transformation or the cumulative number of LPG packages distributed). However, the results are qualitatively similar (not shown).

[16]It covered only five provinces: West Java, East Java, North Sulawesi, Gorontalo, East Nusa Tenggara (NTT).

the rapid growth in the demand during 2007–2012, Pertamina relies on import to secure the supply because building new LPG facilities is costly and time-consuming.[17] Nonetheless, while the program might have triggered other local business to grow, these factors were not directly improved health outcomes.

III.B Infant Mortality

I use three rounds of the Indonesian Demographic and Health Surveys (IDHS), the main source for Indonesia's national health statistics, for the years 2002, 2007, and 2012.[18] Each survey records a cross-section of women aged 15-49 and their basic socio-demographic information, including all of their birth histories. Pregnancy-related variables are recorded only for children born within the last five years.

There are potential sources of bias which can make my estimates serve as a lower bound. First, recall error exists when respondents are more likely to forget distant childbirths or under-reported births when they do not want to talk about the death of their child. This recall error of births may lead to attenuation bias. However, this problem is less serious for recent births rather than more distant births. Therefore, to minimize this problem, I limit my analysis to births within five years preceding the survey and I include dummies for the recall period, following Ngandu et al. (2016). Second, survival bias can exist when the fertility of surviving and non-surviving women differs substantially. Third, mothers who suffer from fetal losses might decide to not become pregnant. Hence, the children in the sample are born from mothers who are generally healthier than those who decided to not become pregnant.

The main analysis is limited to births within five years preceding the survey in order to be able to use pregnancy-related variables and to limit the recall bias. I also assume that all infants are born in the same district as where the mother lived in the survey year.[19]

Table 1 shows the summary statistics of baseline characteristics. Column 1, row 1 shows the average of infant deaths across all targeted districts at baseline, 34 infants death per 1,000 live births, with

[17] There were only a few new LPG storage facilities and refilling stations were built during 2007–2012.

[18] IDHS 2007 and 2012 include all provinces (33 regions and 354 districts) whereas IDHS 2002 excludes 4 regions: Nanggroe Aceh Darussalam, Maluku, North Maluku, and Papua due to the unstable political situation.

[19] In general, mobility across districts in Indonesia is very low (see Figure A.1 in Appendix).

a third of those deaths occurring within the first day after birth. The average of infant death in control districts at baseline is higher than the targeted districts, 40 infants death per 1,000 live births.[20] This is not surprising as the Indonesian government selected the timing of the implementation mainly based on the district's level of kerosene consumption. Districts with a higher level of kerosene consumption are more economically developed compared to those with a lower level of kerosene consumption, and thus those regions are more likely to have a lower infant mortality rate. Therefore, I split the sample in column 1 into (1) early targeted districts that had any LPG distributed during 2007-2008 in column 2 and (2) late targeted districts that had any LPG distributed during 2009-2011 in column 3.[21] As shown in column 3, compared to the early targeted districts, the average of infant deaths in the late targeted districts is more similar with the average of infant death in the control group, 37 infant deaths compared to 40 infants deaths per 1,000 live births.

III.C Time-Variant Household Characteristics

In my most parsimonious model, I use indicators for recall periods, male child, survey periods, and multiple births as the control variables. In my most comprehensive model, I include a full set of control variables which consist of indicators for mother and father education (no education and higher than primary school), mother's age at birth, mothers younger than 18 years old, birth order (second, third, or fourth and higher order), an indicator if parents are smokers, if households have access to clean water sources for drinking (i.e., it is equal to one if households have access to water from protected wells, water pipe built inside the dwellings, bottled water or filtered water, otherwise it is zero), if households have private toilets, if households have access to electricity, if households own a fridge, if households own a TV, and the number of visits to health facilities in the last 12 months. As household characteristics are only recorded at the survey year, I assume that these characteristics do not change within five years preceding the survey.[22]

Balancing checks on covariates. Table 1 shows summary statistics at baseline and Table 2 shows

[20] For comparison, the country's infant mortality rate published from Indonesia Bureau Statistic (BPS) in 2002 and 2007 is 35 and 34 infants death per 1,000 live births, respectively.

[21] IDHS 2012 fieldwork was conducted (from May 7 to July 31, 2012), thus I classified districts that were targeted in 2012 as the untargeted districts.

[22] Note that, with or without controlling for household characteristics, my estimates are very similar. It suggests that this assumption does not play an important role in my analysis.

differential trends of baseline characteristics. Table 1, columns 1 and 4, show that the probability of households using kerosene is 40% and 25% in the treatment group and the control group, respectively. This supports the fact that the timing of the program implementation is correlated with the initial level of kerosene consumption. In the treatment group, the probability of households using LPG is 10%, while it is 4% in the control group.[23] However, health-related variables such as the total visits to health facilities in the last 12 months and the proportion of households who do not smoke, on average, are similar.[24] Households in the targeted districts are more educated and economically better (e.g., they are more likely to own a TV, to own a fridge, to have a private toilet, and to have access to electricity). The number of children born in the last five years is higher for the targeted districts compared to the untargeted districts, but the difference is very small. Comparing the overall level difference, however, households' characteristics in the late targeted districts are more similar to those in the control group (column 3), compared to those in the early targeted districts (column 2).

Parallel trends. Table 2, row 1 shows the pre-implementation trend in infant mortality. The parallel trends in infant mortality reassure my identification strategy explained formally in Section III.D. There are several trend differences in household characteristics at baseline. However, most of these differences are small and work against my findings. For example, the trend difference in the number of children born in the last five years is negative and very small (column 3). Similarly, the trend differences in the ownership of TV and in the availability of toilet in the house (column 3) also work against finding any effect.

The access to clean water for drinking is an important characteristic, considering that one of the leading causes of child mortality in Indonesia is diarrhea. These trends are positive and statistically significant from the control group when I use all targeted districts as the treatment group (column 3). However, the trends are not statistically different from the control group when I use the late targeted districts as the treatment group (column 5). It suggests that the late targeted districts serve as a better counterfactual for the control group in the absence of the program compared to the early targeted districts. Therefore, in my main analysis, I use the late targeted districts as my treatment group in an at-

[23] Before the program, LPG was not subsidized. Also, the LPG cylinder was different with the LPG cylinder designed specifically for the program.

[24] The smoking behavior was recorded at the time of the survey. It is possible that those who are not currently smoking did so in the past. Therefore, the incomplete history of this behavior is one limitation of this data.

tempt to minimize time-varying unobservable factors that can confound my estimates.[25] Additionally, Figure 6 provides a nuanced picture of the trends in infant mortality before and after the program.

III.D Empirical Estimation

From balancing checks shown earlier, it is plausible to assume that the program status is not correlated with any other changes after the program, conditional on district and year fixed effects. Hence, I use a generalized difference-in-differences (DID) model with fixed effects to estimate the causal effect of the program on infant mortality, expressed formally below:[26]

$$Pr(y_{irt} = 1) = c + \alpha_r + \beta_t + \theta Program_{rt} + \varepsilon_{irt} \tag{2}$$

where y_{irt} takes the value of 1 if the infant i in region r at time t is dead within one year and 0 otherwise.[27] α_r and β_t are district and year of birth fixed effects to control for permanent unobserved differences across districts and years. The treatment variable $Program_{rt}$ is either (1) a binary program indicating any LPG distributed in district r starting in the middle of year t[28] or (2) a continuous measure of program intensity in district r at time t (calculated in Equation 1). ε_{irt} is the error term. The intent-to-treat effect (later called program effect) is captured by the θ coefficient.

I include district-specific linear and quadratic trends to account for the yearly trends that were not accounted by district fixed effects.[29]. Additionally, to account for any changes in the observables pre and post-program, I include household and birth characteristics as well as their interaction with a post-program dummy. My most comprehensive specification takes the following form:

$$Pr(y_{irt} = 1) = c + \alpha_r + \beta_t + \theta Program_{rt} + \delta_{rt} + \delta_{rt}^2 + \tau_0 X_{irt} + \tau_1 X_{irt} \text{x} Post_t + \varepsilon_{irt} \tag{3}$$

[25] I show more evidence in Section IV.A to further explain the reason behind the treatment group selection. Additionally, in my further analysis, with alternative subsamples that include all targeted districts, I show that my findings are not driven by treatment group selection.

[26] I use linear probability model for ease of interpretation. Nonetheless, the coefficients from fixed-effects logistic regressions have the same sign, thereby consistent with my results (not shown).

[27] The histogram of infant deaths by the days after death is shown in the Appendix, Figure A.2.

[28] As the month of the program implementation is unknown, I choose the middle of the year (i.e., July) as the start of the program to account for lags in the implementation time.

[29] Indonesia Database for Policy and Economic Research (The World Bank, 2015) provides a panel of district characteristics. However, considering that the data is sparse and only available for a few years, it is not possible to match IDHS with this database without losing many observations

where δ_{rt} and δ_{rt}^2 are district-specific linear and quadratic time trends, respectively. X_{irt} is a set of covariates that captures birth, parental and household characteristics and X_{irt}x$Post_t$ is the interaction of each control variables with a dummy of post-program year, capturing any changes in household characteristics after the program.

IV. RESULTS

First, I present the program impacts on households cooking fuel choice. Second, I present the program impacts on infant mortality with several different specifications. Lastly, I present the program impacts on other birth outcomes.

IV.A Cooking fuel as the main channel

The primary channel through which the program could have an effect on infant mortality is via households' cooking fuel choice.[30] To explore this, I run regressions of the treatment variable on each of the household characteristics. The results show the program is strongly correlated with households' cooking fuel types, and weakly correlated with the other observables. Thereby, it suggests that cooking fuel is likely the main channel.

In Table 3, columns 1-4 use program dummy and columns 5-8 use program intensity as the treatment variable. Each row is a separate regression that includes the treatment variables, district and year fixed effects, as the controls. Columns 1-2 and 5-6 use all samples while columns 3-4 and 7-8 use only late targeted regions. Columns 1, 3, 5, and 7 report the θ coefficient in Equation 2, while columns 2, 4, 6, and 8 report its corresponding standard errors.

There are two important findings from Table 3. First, the program had a high adoption rate. In particular, the program is associated with a 21-40 percentage-point increase in the probability of using LPG and a 18-31 percentage-point decrease in the probability of using kerosene. Second, using program intensity as the treatment variable (columns 5 and 7) almost doubles the probability of using LPG compared to those using only a dummy program (columns 1 and 3). It suggests that a simple binary treatment is insufficient to capture the gradual increase in the access to LPG; therefore, it is a better proxy to reflect the gradual decrease in the dirty fuel consumption compared to the program dummy.

[30] The main cooking fuels in Indonesia are LPG, kerosene, and wood fuel (shown in Figure 3).

As expected, there is no correlation between the treatment variable with the probability of using wood fuel. Households who use wood fuel neither were targeted by the program nor have any incentives to switch to LPG as they can obtain wood fuel for free or with a little cost.

Table 3, column 1 also suggests that the program implementation is associated with both observed and unobserved factors that are also likely to be associated with infant mortality. However, these correlations are overall insignificant and smaller when I use late targeted districts as the treatment group (column 3) compared to those using all targeted districts (column 1). The absence of trend differences between the treatment and the control group on the observable characteristics may suggest that there are no trend differences between the treatment and the control group on the unobservable characteristics. Therefore, these results add support to exclude the early targeted regions.[31]

IV.B The Program Impact on Infant Mortality

Here I present the key results of the paper. I begin by presenting the program impact on infant mortality using a binary program and the program intensity. Then, I show the program effects are overall consistent with different alternative specifications (i.e., within rural areas and within mothers).

Table 4 shows the program impact on mortality using a binary program. Column 1 uses year fixed effects, column 2 adds district fixed effects, column 3 adds household and individual characteristics, column 4 adds district-specific linear trends, column 5 adds district-specific quadratic trends, and column 6 adds all interactions between the control variables and the post-program dummy, cumulatively. Standard errors are clustered by district to allow within district correlations. The coefficient on the program dummy captures the program effect on infant mortality (θ in Equation 3).

The coefficients in the program dummy in all of the specifications indicate that the program is negatively associated with infant mortality. These coefficients are also stable across specifications, even when I add district trends and a full set of control variables. Column 3 replicates the results from Imelda (2018a). Column 6 shows that the program decreased infant mortality rate by 1.2 percentage points. Next, I use my preferred specification in Table 5, which replaces the dummy program with a

[31] Imelda (2018a) provides plausible reasoning why households in the early targeted districts are likely behaving differently due to operational issues of the program implementation. In addition, using only late targeted regions removed mostly regions in Java. Central Java experienced eruptions of Mount Merapi in 2010 (Damby et al., 2013). It was the largest since the 1870s, and led to evacuations of 320,000 people from the affected area. It emitted toxic volcanic ash particles surrounding Java and may contribute a differential trend in infant mortality.

continuous variable of program intensity.

The program impact on infant mortality using program intensity is presented in Table 5. Similar to the earlier results with a binary program, the program is negatively correlated with infant mortality. Column 5 indicates that a 10-percentage-point increase in the free initial LPG distributed decreases the probability of infant death by 3.3 percentage points. However, some trends in the program intensity are accounted in the district trends.[32] Hence when I add linear and quadratic time trends of districts, the program intensity coefficient is smaller and no longer statistically significant, unlike the estimates using program dummy. The coefficient of household characteristics are consistent with the literature (e.g., more educated parents have lower infant mortality). The inclusion of a full set of controls (column 4) gives similar estimates as only including year and district fixed effects (column 3), suggesting that the program effects cannot be explained by the observables.

The estimates using program intensity (Table 5) are precise and consistent with the estimates using program dummy (Table 4). Using program dummy, the program led to 103 infant deaths averted from 9,420 live births observed in the late targeted districts after the program. On average, there are 28 LPG packages distributed per 100 people in the late targeted districts. Therefore, using program intensity, the program prevented approximately 87 infant deaths in total. This is slightly lower than previous estimates with program dummy, which is expected considering that the program dummy may overestimate the effect due to the imprecise measurement of the program.

In the event study plot, I provide a graphical analysis of the policy effect on infant mortality in Figure 7. It shows the program intensity coefficient θ in Equation 2 by years since program implementation. These estimates are equivalent to the specification in Table 5, column 3. Each coefficient represents the mean difference between the treatment group and the control group, using the year 0 since the program implementation as the reference category.

The event study analysis complements the earlier results in two ways. First, it provides graphical evidence of the parallel trend. The coefficients of the program intensity before the implementation year are essentially zero, reassuring the parallel pre-trend showed earlier (Table 2, column 5, row 4). Second, it reflects the magnitude of the program impact over time. The program reduced infant mortality rate by 3 percentage points within the first year of the program, and the effects were doubled

[32] When I regress district trends on program intensity, the R-squared is about 0.27.

in the second year of the program.

Comparing to the existing literature, these estimates are within the range of the health effects estimated in the ambient air pollution studies in developing countries.[33] For example, Jayachandran (2009) finds 15,600 "missing children" due to massive forest fires in Indonesia; Tanaka (2015) finds that air regulation in China reduces three infants death per 1,000 births, Cesur et al. (2016) finds that a one-percentage-point increase in natural gas subscriptions in Turkey decrease infant mortality by 4%; Arceo et al. (2016) find a 1% increase in PM_{10} over a year leads to a 0.40% increase in infant mortality, while a 1% increase in CO results in a 0.33% increase; Greenstone and Hanna (2014) find that a policy on reducing vehicular pollution in India is associated with a reduction in the infant mortality rate of 0.64 per 1,000 live births. Also, my estimates are within the range of the Chay and Greenstone (2003) study in the United States context, which finds a lower infant mortality rate by 3-6 percent due to the nonattainment status in 1972.[34]

My findings are parallel with several indoor air pollution studies that focus on different outcomes and studies that use different research design. For example, Barron and Torero (2017) find that the randomized encouragement to connect to an electrical grid led to up to a 14 percentage-point lower prevalence of acute respiratory infections among children under six; Pillarisetti et al. (2017) find 44,000 overall deaths averted over five years using a hypothetical policy simulation of full LPG adoption in India; Andadari et al. (2014) study the same program and find that it effectively improved access to modern energy among the poorest households.

Within rural areas estimates. Within rural area[35] estimates can be a simple way to remove potentially omitted variables from ambient air pollution (i.e., from traffic congestion), which has been shown to have severe health consequences for infants (Currie and Walker, 2011; Knittel et al., 2016). Unfortunately, a reliable measurement of ambient air pollution for the whole nation is nonexistent. However, intuitively, more urbanized areas are likely to have higher ambient air pollution (i.e., higher local air pollution from the transportation sector), compared to rural area. For example, Resosudarmo

[33]The estimates of ambient air pollution in developing countries are usually much higher than those in developed countries.

[34]Note that due to the lack of indoor air quality measurements, my estimates do not imply differences in the marginal effect of indoor air quality.

[35]An area is classified as rural based on a scoring system. For example, if the regions have a population density of fewer than 5,000 persons per square kilometer and if the percentage of household working in agriculture sector is higher than 25%. There are other categories for the scoring system such as the number of school, health care, roads, etc (Mulyana, 2014).

and Napitupulu (2004) find a positive correlation on ambient air pollution and health costs in Jakarta, the capital city of Indonesia which has some of the worst traffic congestion in the world (Fickling, 2018).

Using all observations in the rural area, including all targeted districts, I redo the same regression in Equation 3 to estimate the program effects on the infant mortality as the treatment group and all the untargeted districts as the control group. The results are shown in Table 6, columns 1-5. Panel 1 shows the program impacts using program dummy (similar to Table 4) and Panel 2 shows the program impacts using program intensity (similar to Table 5).

The result in column 5 is consistent with the earlier findings (i.e., a ten percentage-point increase in the program intensity would lower the infant mortality by 3.4 percentage points). The estimates using program intensity are precise even after accounting for district trends, unlike those using program dummy (Panel 1). Again, it suggests that the increasing access to LPG in the rural areas is not well captured by the dummy program. Compared to the estimates in Table 5, these effects are very similar, suggesting that the program effects are not driven by subsample selections.

Within mother estimates. To control for unobservable time-invariant characteristics of mothers, I use mother fixed effects on the sample of mothers with more than one birth. Table 6, column 6 shows the results that include mother fixed effects. I assume that all infants are born in the same district as where the mother's lived in the survey year. As expected, there are not many births within one mother, leading to larger standard errors. However, the sign and the magnitude of the point estimates are very similar with my earlier estimates, indicating that the program impact on infant mortality cannot be explained by unobservable fixed characteristics of mothers.

IV.C The Program Impact on Birth Outcomes

Finally, I aim to investigate if the indoor air pollution is disproportionately affecting fetuses and the newborns by exploring the effect on deaths occurring at different time periods and on birth weight. Low birth weight children and very low birth weight children are often associated with fetal exposure to pollution during pregnancy (for example see Currie 2011).[36]

Table 7 summarizes the results. Each column is a separate regression of program intensity on a

[36] I do not include premature birth and stillbirth due to many missing values.

dummy variable of infant deaths at age 0–1 days (column 1), infant deaths at age 2–30 days (column 2), infant deaths at ages 1-11 months (column 3), infants born with weight less than 2.5 kilograms (column 4), and infants born with weight less than 1.5 kilograms (column 5). All regressions use the richest specification that includes year and district fixed effects, district-specific linear and quadratic trend, full control variables discussed in Section III.C and all of the interactions between the control variables and the post-program dummy. The mean of each outcome variables is measured at baseline and per 1,000 live births.

The results in columns 1-3 on mortality consistently show that the program reduced neonatal and post-neonatal mortality rates. Similarly, the results in columns 4 and 5 indicate that the program reduces the prevalence of low birth weights. However, the estimates are statistically significant for infant deaths within the first day after birth, suggesting that the kerosene-related pollutants may affect fetuses through maternal exposure to pollution. This is consistent with the result in column 5, which also shows that the program is negatively associated with the prevalence of very low birth weight. However, the point estimates in columns 2, 3, and 4 suggest that the earlier estimates on infant mortality are not driven by the reduction in infant deaths occurring after the first day of birth and not driven by the prevalence of low birth weights.

V. Mechanism

There are various channels for which the program could impact infant mortality. The most salient is through program induced improvement in indoor air quality. However, there are other possible channels such as changes in expenditure, fuel stacking, and avoidance behavior, which I discuss in this section. Overall, I show that that the improvement in indoor air quality played a major role.

V.A Indoor Air Quality

Exposure studies that compare emissions from kerosene and LPG stoves support that kerosene-using stoves are more polluting compared to LPG stoves. Unfortunately, there is no credible indoor air quality measurement that covers a whole country. However, to shed light on this mechanism, I briefly discuss the exposure studies that investigate the relative emission of kerosene-using stoves and LPG-using stoves. Further, I discuss how the program is highly correlated with the reduction in kerosene consumption, and so associated with an improvement in indoor air quality.

Switching from kerosene stove to LPG stove is associated with a 30-percent reduction in $PM_{2.5}$ personal exposure (Andresen et al., 2005)[37], illustrated in Figure 8 and a 20-percent reduction in CO concentration (Smith et al., 1999)[38]. These claims are also consistent with Smith et al. (2000), who provide comparison emission level per-meal by each fuel type.[39] $PM_{2.5}$ and CO are some of the byproducts of incomplete combustion from cooking fuels that are believed to have negative effects on infants, both through prenatal exposure and after birth exposure.[40] In contrast, burning LPG produces significantly less NO_x, CO, and small amounts of SO_2 (EPA). Note that most of these studies exclude any spillover effects from neighborhood emissions (i.e., indoor air pollution from neighbors that contribute to local outdoor air pollution), thereby the improvement in indoor air quality from kerosene to LPG switching can be larger.

The program is strongly correlated with a substantial reduction in kerosene consumption.[41] Firstly, as discussed earlier in Figure 3, the average reduction in kerosene shares is very similar to the average reduction in LPG shares at the district level. Hence, it gives reassurance that program intensity serves as a good proxy for the reduction in the kerosene consumption. Furthermore, using a longitudinal survey of the Indonesian Family Life Survey (IFLS), Imelda (2018b) shows that households reduce their kerosene consumption down to zero after the program.[42] For such a big reduction in the kerosene consumption within a short period of time, plus the improvement in indoor air quality associated with the fuel-switching, the reduction in infant mortality is likely to have been attributed, at least partly, to the improvement in indoor air quality induced by the reduction in the kerosene consumption.

I find no effects on infant mortality among kerosene users and among households who do not have access to electricity, shown in Table 8. Households who do not have access to electricity were likely not affected by the program because they could still rely on kerosene as their source of lighting. These

[37] Andresen et al. (2005) provide 24-hour personal exposure and indoor concentration of $PM_{2.5}$ from kerosene and LPG stoves in Mysore, a medium-scale city in India where kerosene and LPG were the primary domestic fuels. During summertime, among 30 women in the study, the arithmetic mean of personal exposure for kerosene users was 148 $\mu g\,m^{-3}$ (with standard error (SD) of 11 $\mu g\,m^{-3}$) and for LPG users was 71 $\mu g\,m^{-3}$ (SD 15 $\mu g\,m^{-3}$).

[38] Using a simulated kitchen built in India, Smith et al. (1999) provide a database containing a set of emissions from 28 types of stoves and fuel commonly used in developing countries.

[39] In terms of stove variety, anecdotally, there is only one type of LPG stove distributed throughout the program, therefore the type of kerosene stove and LPG stove does not vary across households in general.

[40] Other pollutants such as nitrogen dioxide (NO_2) and sulfur dioxide (SO_2) surrounding the kitchen when kerosene is used are also far above WHO standards (Smith et al., 2005; Apple et al., 2010; Hasanudin et al., 2011; Lam et al., 2012; Adeniji et al., 2015; Maiyoh et al., 2015).

[41] Ideally, I could do a regression of program intensity on households kerosene consumption. However, this is not possible as IDHS does not record households' fuel consumption.

[42] Imelda (2018b) uses the kerosene quantity on the last purchase on the month before the survey as a proxy for kerosene consumption.

results serve as placebo checks, mitigating concerns that the program intensity is confounded with unobservable determinants of infant mortality.

V.B Other Channels

Expenditure channel. The reduction in infant mortality can be driven by program-induced health investment. LPG is more cost efficient compared to kerosene, approximately 0.4 kilograms of LPG can replace one liter of kerosene (Budya and Arofat, 2011). Households who switch from kerosene to LPG experienced a 40% reduction in fuel expenses or about 1.19 USD per month, but no changes in other non-durable expenditures (Imelda, 2018b). This is intuitive as the changes in the fuel expenses are small, 2% of the total monthly expenditure. Similarly, Budya and Arofat (2011) report several surveys of the program which indicate a 30% reduction in their expenditure on cooking fuel due to the program, 1.64 USD on average in monthly savings.

This expenditure channel, however, is unlikely to be the dominating channel in which the program could affect infant mortality. These extra savings due to the program, are not only very small but also are not necessarily spent on health-related investment (e.g., the extra monies can be spent on healthy food, but also can be spent on cigarettes). Even the conditional cash transfer in Indonesia, amounting up to 20% of *total consumption* of the poor households, did not lead to any significant reduction in infant mortality (Alatas et al., 2011).

Fuel stacking. Households tend to use a mix of fuels that are available within their locations, subject to their budget constraints. Hence, I do not use this categorical variable because they are subjected not only to endogeneity but also to measurement error. Therefore, any presence of fuel stacking is included in my estimates. In particular, the estimated program effects include the effects from the switch between not only kerosene to LPG but also wood to LPG.[43] However, Table 3, row 3 shows that the program has no effect on households' probability of using wood fuel as their *primary* cooking fuel, thereby it is not likely to play a major role.[44] Thereby, any program effect captured in the estimates is

[43] The program eligibility is based on whether households have ever used LPG. Consider when households report kerosene as their *primary* cooking fuel, thus they are eligible for the program. Although, in fact, they use mixed fuels, a combination of wood fuel and kerosene.

[44] Two other studies that investigate the impact of this program, support that fuel stacking likely disappeared after the program. Based on a survey conducted on 550 households in five sub-districts in Central Java, Andadari et al. (2014) find no impact on the number of households that use only wood fuel for cooking. Before the program, all households consumed some kerosene, despite cooking fuel they reported, Imelda (2018b). However, after the program, kerosene consumption among all households fell to zero, while kerosene consumption among households who report using kerosene as their primary cooking fuel does not change.

likely driven mostly by the reduction in kerosene consumption despite their true cooking fuel mix.

Avoidance behavior. The literature on ambient air pollution and health suggests that optimizing individuals might minimize their exposure to protect their health. Similarly, avoidance behavior can contribute to a reduction in indoor air pollution exposure. However, I see no compelling reason to believe that the avoidance behavior is systematically correlated with the program.

When outdoor air quality is bad, individuals can stay indoors, but when indoor air quality is bad, individuals have limited options to minimize the indoor air pollutant exposure. It is possible that when households use their kerosene stoves, they tend to open their windows, but when they use LPG stoves, they do not feel the need to open the windows to increase the ventilation. However, studies on how effective ventilation can improve health are inconclusive. For example, Dasgupta et al. (2004) find that the location of a kitchen has large and statistically significant effects on 24-hour average indoor pollutants concentration, but Pitt et al. find that an increase in the permeability of roofs or walls has no significant effect on health. Plus, Saksena et al. (2003) show that households who use kerosene exhibit limited avoidance behavior compared to when they use biomass. They find that households who use kerosene cook more often and closer to the kerosene stoves. As smoke from kerosene stoves is less visible, and switching from kerosene to LPG likely does not have noticeable differences in indoor smoke, thereby avoidance behavior, if any, is likely to be small.[45]

VI. Robustness Checks and Heterogeneous Effects

VI.A Robustness Checks

In the earlier sections, I show that my estimates are robust with or without the inclusion of the household characteristics, district-specific trends, and the post-program observables trends. I also show that the results are consistent under alternative subsamples, thus supporting my causal claim. Further robustness checks are presented in Table 9. All outcome variables are infant mortality. Each column is a different panel based on different specifications as follow.

In column 1, I restrict the sample to households that have similar observed characteristics. I first compute the propensity scores of program intensity based on the household characteristics used as the

[45] Especially poor households likely do not own any indoor air purifiers. They even more likely to not be aware of the adverse health risks of indoor air pollution.

control variables. Then, I re-estimate the program effects using observations only under the common support. In column 2, I cluster the standard error at the household level. In column 3, I add month-year of birth fixed effects to account any variation due to seasonality (e.g., when households work in an agriculture sector, seasonality might influence infant mortality, since their income often rises during certain months and seasons). In column 4, I account for the number of household members, considering that bigger households consume more cooking fuel.[46] In column 5, I account for the number of children born within the last five years preceding the survey, since infant mortality can decrease simply because there are more births within a household.[47] In column 6, I use 2004 as a placebo cut-off year. Hence, births after 2004 in the targeted districts are defined as treatment groups.

The results in columns 1-5 suggest that the estimated coefficients are unchanged even after using propensity score matching, changing standard error clustering, the inclusion of month-year fixed effects, number of household members, children born in the last five years. Similarly, there is no effect of placebo treatment on infant mortality which indicates that trends in infant mortality are similar in the pre-program period.

Lastly, the reduction in infant mortality might be driven by selective migration. It is difficult to find any compelling reason that the program is correlated with selective migration. I expect that the program is not a strong driver for a household to migrate to a different district considering the cost of moving. In general, mobility across districts in Indonesia is very low. While IDHS does not record any migration history, I show that migration across districts involves less than 5% of from the total population. The graphical evidence, using Indonesia census of 2000, 2005 and 2010, is shown in Figure A.1 in Appendix.

VI.B Heterogenous Effects

In this section, I explore the heterogeneous effects from subsamples based on program duration, mothers' education, poverty status, and child's gender shown in Table 10. All of the regressions use infant mortality as the dependent variable and a full set of controls: year fixed effects, district fixed effects, district-specific linear trends, district-specific quadratic trends, control variables (discussed in Section

[46]It was excluded in my earlier analysis to exclude potential correlation with infant mortality.

[47]Similar to column 4, this variable is excluded from the main analysis as it is correlated with the number of infant deaths.

III.C) and the interactions between control variables and the post-program dummy.

Panel 1: Effect by program duration. The effect of the program intensity might be accumulated in the mother's health. Longer exposure to the program may be associated with healthier mothers, thus they will likely to have healthier babies compared to mothers with shorter exposure to the program. Thus, I separately estimate the effects by the implementation year.[48] The result indicates that the effects are larger among those exposed earlier, suggesting the potential "accumulated effects" on mothers.

Panel 2: Mother's education. A large body of literature has supported that low educated and poor households are the most vulnerable group to environmental risk. In Panel 2, the results suggest that the program effects are larger among mothers that had relatively higher education. A possible explanation is that mothers with low education already have a low health endowment, so a marginal improvement in the indoor air quality induced by the program is insufficient to reduce infant mortality. Alternatively, mothers with higher education tend to be supported by other factors such as access to health facilities and better birth attendance, so the program is sufficient to reduce infant mortality among them.

Panel 3: Effects among the poor. The poor category is constructed based on whether or not households own a private toilet. This category takes value one if households own a private toilet with a septic tank, and zero otherwise. It takes value zero if households use a private toilet without a septic tank, public toilet, pit latrine, bush, river, composting toilet, and other. This is a simple category that can reflect the true condition of households' economic condition. The results suggest the program effect on infant mortality is larger among poor households.

Panel 4: Effects by gender. The program effects on infant mortality are larger among girls. This is of great importance in developing countries, where gender discrimination often exists. The benefits of the indoor air quality improvement can be enjoyed by both genders equally. However, female fetuses may have a higher threshold at which pollution leads to mortality than boys do. This is consistent with a large body of literature regarding boys being biologically weaker and more susceptible to environmental risks.

[48] There are only a few districts targeted in 2010, thus I combine them with district targeted in 2011.

VII. Conclusions

The seventh United Nation Sustainable Development Goal calls for universal access to affordable, reliable, sustainable and modern energy by 2030. There are about 2.3 billion people globally who do not have access to clean cooking fuels (The World Bank, 2018). Interventions in clean energy need to be scaled up significantly to support this agenda and proper cost and benefit calculations are needed urgently. Despite its importance, to my knowledge, there is no study that measures the causal health impact from a large-scale household fuel conversion program. Traditional cost and benefit analyses often leave out the health benefits in the calculation as there are no reliable estimates of the health benefits of adopting clean energy, thereby underestimating the total benefits.

I estimate the health impact of indoor air pollution using a nationwide fuel-switching program in Indonesia. The program reached more than 50 million homes and successfully reduced kerosene consumption by more than 80% within four years. I find that the program led to a significant reduction in infant mortality. In particular, a 10-percentage-point increase in the free initial LPG distributed reduces the infant mortality by 3.3 percentage points or 1.2 infants per 1,000 live births. Program-induced indoor air quality improvement likely played an important role since switching from kerosene to LPG is associated with a substantial reduction in the average exposure of $PM_{2.5}$ and CO (Andresen et al., 2005). This study provides the lower-bound estimates on the health benefits of switching to cleaner cooking fuel. If moving away from kerosene towards LPG led to a significant reduction in infant mortality, then moving away from biomass (i.e., the dirtiest fuel) is expected to lead to even greater health benefits.

Using some back of the envelope calculations, I estimate that the program leads to approximately 1,687 fewer infant deaths on every ten-percentage-point increase in the program intensity, in 2011. This calculation is based on the total live births of around 1.6 million.[49] The benefits from these avoided deaths yield an estimated USD 422 million in annual savings.[50] On the cost side, even without accounting for any health benefits, the program already had a net saving of about USD 2.9 billion from the removal of kerosene subsidy (Budya and Arofat, 2011).

[49] Total live births are calculated from a total population in the treatment group of 87 million and a crude birth rate of 19.2.

[50] I use a value of statistical life in rural Thailand, USD 250,000 (Gibson et al., 2007), which is likely closer to rural Indonesia.

The current investigation is particularly timely as estimating the link between kerosene use and health has become increasingly important. First and foremost, this paper supports the recent World Health Organization (WHO) guidelines that discourage the use of kerosene. The WHO recently reclassified kerosene as a 'dirty fuel' for the first time (WHO, 2016), putting it in the same class as biomass.[51] Furthermore, given that a large population is still using kerosene, the global health impact of moving away from it is likely quite large. Kerosene still plays a major role in household consumption in most developing countries. Globally, approximately 500 million households use kerosene for cooking (Lam et al., 2012) and one billion people rely on kerosene and other polluting devices for lighting (WHO, 2016). Most developing countries continue to encourage the use of kerosene by highly subsidizing it. The average direct subsidies of kerosene reach approximately $44 million per day (Mills, 2017). Not only is the cost of subsidizing kerosene high, but the health costs of using are also likely to be substantial.

The total health benefits of the program are likely to be much greater than documented since this paper only focuses on one of the possible health benefits of the program, infant mortality. Other health benefits, such as fewer respiratory illnesses, improved child and adult morbidities, and other benefits outside of health such as labor force participation and environmental impact, are left for future research. For example, Budya and Arofat (2011) discuss that the new investment in LPG created 28 thousand new jobs and reduced CO_2 emissions by 8.4 million tonnes per year. A full accounting of the different ways that indoor air pollution affects health is beyond the scope of this paper, but the findings from this paper suggest that the health benefit of providing a cleaner fuel is quite large and significant among infants, the most vulnerable group.

UNIVERSIDAD CARLOS III DE MADRID

[51] In the past, kerosene was grouped as a clean fuel together with gas and electricity.

References

Deborah H Bennett, Thomas E McKone, John S Evans, William W Nazaroff, Manuele D Margni, Olivier Jolliet, and Kirk R Smith. Defining Intake Fraction, 2002.

Marta Oliveira, Klara Slezakova, Joana Madureira, Eduardo de Oliveira Fernandes, Cristina Delerue-Matos, Simone Morais, and Maria do Carmo Pereira. Polycyclic Aromatic Hydrocarbons in Primary School Environments: Levels and Potential Risks. *Science of The Total Environment*, 575:1156–1167, 2017.

Weeberb J Requia, Matthew D Adams, Altaf Arain, and Mark Ferguson. Particulate Matter Intake Fractions for Vehicular Emissions at Elementary Schools in Hamilton, Canada: An Assessment of Outdoor and Indoor Exposure. *Air Quality, Atmosphere & Health*, 10(10):1259–1267, 2017.

IHME. Institute of Health Metrics and Evaluation - Global Burden of Disease (GBDx) Data Tool. 2016. URL `http://ghdx.healthdata.org/gbd-results-tool`.

Esther Duflo, Michael Greenstone, and Rema Hanna. Indoor Air Pollution, Health and Economic Well-Being. *SAPI EN. S. Surveys and Perspectives Integrating Environment and Society*, (1.1), 2008.

William J Martin, Roger I Glass, John M Balbus, and Francis S Collins. A Major Environmental Cause of Death. *Science*, 334(6053):180–181, 2011.

S Semple, C Garden, M Coggins, KS Galea, P Whelan, H Cowie, A Sánchez-Jiménez, PS Thorne, JF Hurley, and JG Ayres. Contribution of Solid Fuel, Gas Combustion, or Tobacco Smoke to Indoor Air Pollutant Concentrations in Irish and Scottish Homes. *Indoor air*, 22(3):212–223, 2012.

Kenneth M Bryden, Nigel Bruce, and Michael Peck. WHO Indoor Air Quality Guidelines: Household Fuel Combustion – Review 5: Population Levels of HAP and Exposure. *Geneva: World Health Organization*, 2015.

Kenneth Y Chay and Michael Greenstone. The Impact of Air Pollution on Infant Mortality: Evidence from Geographic Variation in Pollution Shocks Induced by a Recession. *The quarterly journal of economics*, 118(3):1121–1167, 2003.

Janet Currie and Matthew Neidell. Air Pollution and Infant Health: What Can We Learn from California's Recent Experience? . *The Quarterly Journal of Economics*, 120(3):1003–1030, 2005.

Janet Currie and Reed Walker. Traffic Congestion and Infant Health: Evidence from E-Zpass. *American Economic Journal: Applied Economics*, 3(1):65–90, 2011.

Rema Hanna, Esther Duflo, and Michael Greenstone. Up in Smoke: The Influence of Household Behavior on the Long-Run Impact of Improved Cooking Stoves. *American Economic Journal: Economic Policy*, 8(1):80–114, 2016.

Lianne Sheppard, Richard T Burnett, Adam A Szpiro, Sun-Young Kim, Michael Jerrett, C Arden Pope, and Bert Brunekreef. Confounding and Exposure Measurement Error in Air Pollution Epidemiology. *Air Quality, Atmosphere & Health*, 5(2):203–216, 2012.

Michael Greenstone and Ted Gayer. Quasi-experimental and Experimental Approaches to Environmental Economics. *Journal of Environmental Economics and Management*, 57(1):21–44, 2009.

PT Pertamina (Persero) WLPGA. Kerosene to LP Gas Conversion Programme in Indonesia. 2012. URL `https://www.wlpga.org/wp-content/uploads/2015/09/kerosene-to-lp-gas-conversion-programme-in-indonesia.pdf`.

Hanung Budya and Muhammad Yasir Arofat. Providing Cleaner Energy Access in Indonesia Through the Megaproject of Kerosene Conversion to LPG. *Energy Policy*, 39(12):7575–7586, 2011.

Penny Rechkemmer Andresen, Gurumurthy Ramachandran, Pramod Pai, and Andrew Maynard. Women's Personal and Indoor Exposures to PM2.5 in Mysore, India: Impact of Domestic Fuel Usage. *Atmospheric Environment*, 39(30):5500–5508, 2005.

KR Smith, R Uma, VVN Kishore, K Lata, V Joshi, J Zhang, RA Rasmussen, and MAK Khalil. Greenhouse Gases from Small-Scale Combustion in Developing Countries: Household Stoves in India. *Research Triangle Park, NC: US Environmental Protection Agency*, 1999.

Michael Greenstone and B Kelsey Jack. Envirodevonomics: A Research Agenda for an Emerging Field. *Journal of Economic Literature*, 53(1):5–42, 2015.

Mark M Pitt, Mark R Rosenzweig, and Md Nazmul Hassan. Sharing The Burden of Disease: Gender, the Household Division of Labor and the Health Effects of Indoor Air Pollution in Bangladesh and India. In *Stanford Institute for Theoretical Economics Summer Workshop, volume=202, year=2006*.

Ani Rudra Silwal and Andy McKay. The Impact of Cooking with Firewood on Respiratory Health: Evidence from Indonesia. *The Journal of Development Studies*, 51(12):1619–1633, 2015.

Imelda. Indoor Air Pollution and Infant Mortality: A New Approach. In *AEA Papers and Proceedings*, volume 108, pages 416–421. American Economic Association 2014 Broadway, Suite 305, Nashville, TN 37203, 2018a.

Kirk R Smith and Ambuj Sagar. Making The Clean Available: Escaping India's Chulha Trap. *Energy Policy*, 75:410–414, 2014.

Ahmed Mushfiq Mobarak, Puneet Dwivedi, Robert Bailis, Lynn Hildemann, and Grant Miller. Low Demand for Nontraditional Cookstove Technologies. *Proceedings of the National Academy of Sciences*, 109(27):10815–10820, 2012.

David Kimemia and Ashley Van Niekerk. Cookstove Options for Safety and Health: Comparative Analysis of Technological and Usability Attributes. *Energy Policy*, 105:451–457, 2017.

Precious Benjamin Meixi Gan Elisa Puzzolo Thoday, Katherine. The Mega Conversion Program from Kerosene to LPG in Indonesia: Lessons Learned and Recommendations for Future Clean Cooking Energy Expansion. *Energy for Sustainable Development*, 2018. URL `https://doi.org/10.1016/j.esd.2018.05.011`.

Roos Kities Andadari, Peter Mulder, and Piet Rietveld. Energy Poverty Reduction By Fuel Switching. Impact Evaluation of the LPG Conversion Program in Indonesia. *Energy Policy*, 66:436–449, 2014.

Robert Sparrow, Jossy P Moeis, Arie Damayanti, and Yulia Herawati. Conditional Cash Transfers in Indonesia: Baseline Survey Report Program Keluarga Harapan and PNPM Generasi. *World Bank. Washington, DC, June*, 2008.

Vivi Alatas, Nur Cahyadi, Elisabeth Ekasari, Sarah Harmoun, Budi Hidayat, Edgar Janz, Jon Jellema, et al. Program Keluarga Harapan: Main Findings from The Impact Evaluation of Indonesia's Pilot Household Conditional Cash Transfer Program. *New York: World Bank*, 2011.

Margaret Triyana. Do Health Care Providers Respond to Demand-Side Incentives? Evidence from Indonesia. *American Economic Journal: Economic Policy*, 8(4):255–88, 2016.

Indonesia Demographic and Health Survey 2002-2003 [Dataset].

Indonesia Bureau of Statistics IDHS. Indonesia Demographic and Health Survey 2007 [Dataset], 2008.

Indonesia Bureau of Statistics IDHS. Indonesia Demographic and Health Survey 2012 [Dataset], 2012.

Nobubelo K Ngandu, Samuel Manda, Donela Besada, Sarah Rohde, Nicholas P Oliphant, and Tanya Doherty. Does Adjusting for Recall in Trend Analysis Affect Coverage Estimates for Maternal and Child Health Indicators? An Analysis of DHS and MICS Survey Data. *Global health action*, 9(1):32408, 2016.

The World Bank. INDO-DAPOER, 2015. URL `https://data.worldbank.org/data-catalog/indonesia-database-for-policy-and-economic-research`.

DE Damby, CJ Horwell, PJ Baxter, P Delmelle, K Donaldson, C Dunster, B Fubini, FA Murphy, C Nattrass, S Sweeney, et al. The Respiratory Health Hazard of Tephra from the 2010 Centennial Eruption of Merapi with Implications for Occupational Mining of Deposits. *Journal of Volcanology and Geothermal Research*, 261:376–387, 2013.

Seema Jayachandran. Air Quality and Early-Life Mortality Evidence from Indonesia's Wildfires. *Journal of Human Resources*, 44(4):916–954, 2009.

Shinsuke Tanaka. Environmental Regulations on Air Pollution in China and Their Impact on Infant Mortality. *Journal of Health Economics*, 42:90–103, 2015.

Resul Cesur, Erdal Tekin, and Aydogan Ulker. Air Pollution and Infant Mortality: Evidence from The Expansion of Natural Gas Infrastructure. *The Economic Journal*, 127(600):330–362, 2016.

Eva Arceo, Rema Hanna, and Paulina Oliva. Does The Effect of Pollution on Infant Mortality Differ Between Developing and Developed Countries? Evidence from Mexico City. *The Economic Journal*, 126(591):257–280, 2016.

Michael Greenstone and Rema Hanna. Environmental Regulations, Air and Water Pollution, and Infant Mortality in India. *The American Economic Review*, 104(10):3038–3072, 2014.

Manuel Barron and Maximo Torero. Household Electrification and Indoor Air Pollution. *Journal of Environmental Economics and Management*, 2017.

Ajay Pillarisetti, Dean T Jamison, and Kirk R Smith. Household Energy Interventions and Health and Finances in Haryana, India: An Extended Cost-Effectiveness Analysis. *Injury Prevention and Environmental Health*, page 223, 2017.

W Mulyana. Rural-Urban Linkages: Indonesia Case Study. *Urban and Regional Development Institute (URDI) J, Indonesia, editor. Santiago, Chile: RIMISP*, 2014.

Christopher R Knittel, Douglas L Miller, and Nicholas J Sanders. Caution, Drivers! Children Present: Traffic, Pollution, and Infant Health. *Review of Economics and Statistics*, 98(2):350–366, 2016.

Budy P Resosudarmo and Lucentezza Napitupulu. Health and Economic Impact of Air Pollution in Jakarta. *Economic Record*, 80(s1):S65–S75, 2004.

David Fickling. We're Running Out of Road, 2018. URL `https://www.bloomberg.com/opinion/articles/2018-11-07/think-new-york-traffic-is-bad-try-bangkok-or-jakarta`.

Janet Currie. Inequality at Birth: Some Causes and Consequences. *American Economic Review*, 101 (3):1–22, 2011.

Kirk R Smith, R Uma, VVN Kishore, Junfeng Zhang, V Joshi, and MAK Khalil. Greenhouse Implications of Household Stoves: An Analysis for India. *Annual Review of Energy and the Environment*, 25(1):741–763, 2000.

Kirk R Smith, Jamesine Rogers, and Shannon C Cowlin. *Household Fuels and Ill-Health in Developing Countries: What Improvements Can Be Brought by LP Gas?* World LP Gas Association Paris, France, 2005.

J Apple, R Vicente, A Yarberry, N Lohse, E Mills, A Jacobson, and D Poppendieck. Characterization of Particulate Matter Size Distributions and Indoor Concentrations from Kerosene and Diesel Lamps. *Indoor air*, 20(5):399–411, 2010.

U Hasanudin, A Haryanto, and J Romero. Effect of Stove Types on in-Kitchen Air Quality: Case Study at Way Isem Village, Lampung Province, Indonesia. *Journal of Sustainable Energy & Environment*, 2(4):181–186, 2011.

Nicholas L Lam, Kirk R Smith, Alison Gauthier, and Michael N Bates. Kerosene: A Review of Household Uses and Their Hazards in Low-and Middle-Income Countries. *Journal of Toxicology and Environmental Health, Part B*, 15(6):396–432, 2012.

BA Adeniji, GREE Ana, BO Adedokun, and OI Ige. Exposure to Emissions from Kerosene Cooking Stoves and the Pulmonary Health Status of Women in Olorunda Community, Ibadan, Nigeria. *Journal of Environmental Protection*, 6(05):435, 2015.

Geoffrey K Maiyoh, Rachel W Njoroge, and Vivian C Tuei. Effects and Mechanisms of Kerosene Use-Related Toxicity. *Environmental toxicology and pharmacology*, 40(1):57–70, 2015.

US Environmental Protection Agency EPA. Ap42 section 1.5 liquefied petroleum gas combustion.

Imelda. The Response of Consumption to Fuel Switching: Panel Data Estimates, 2018b. URL `http://hdl.handle.net/10016/27653`.

Susmita Dasgupta et al. *Indoor Air Quality for Poor Families: New Evidence from Bangladesh*, volume 3393. World Bank Publications, 2004.

Sumeet Saksena, PB Singh, Raj Kumar Prasad, Rakesh Prasad, Preeti Malhotra, Veena Joshi, and RS Patil. Exposure of Infants to Outdoor and Indoor Air Pollution in Low-Income Urban Areas–A Case Study of Delhi. *Journal of Exposure Science and Environmental Epidemiology*, 13(3):219, 2003.

The World Bank. 2018 SDG7 Tracking: The Energy Progress Report. 2018. URL `http://www.worldbank.org/en/news/press-release/2018/05/02/sustainable-development-goal-sdg-7-global-progress-report`.

John Gibson, Sandra Barns, Michael Cameron, Steven Lim, Frank Scrimgeour, and John Tressler. The Value of Statistical Life and The Economics of Landmine Clearance in Developing Countries. *World Development*, 35(3):512–531, 2007.

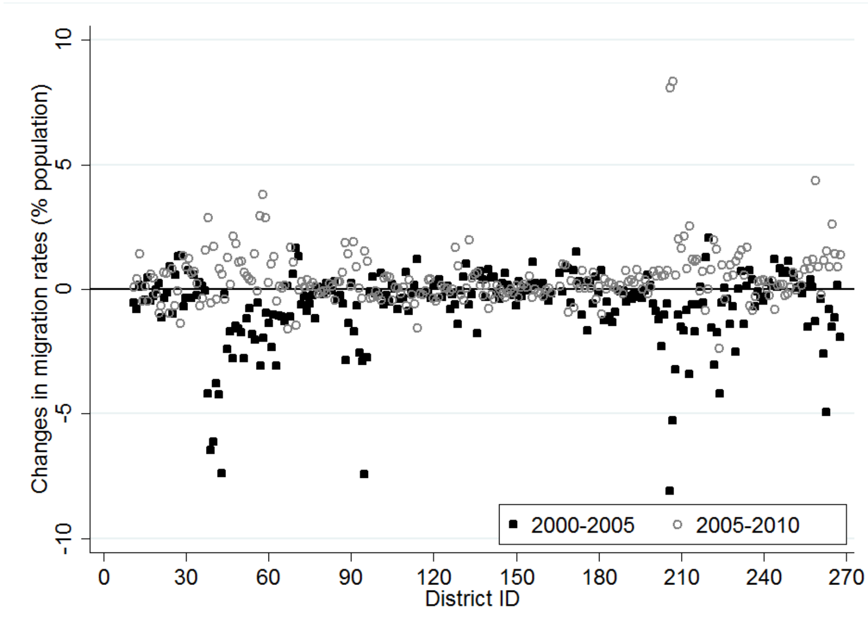
WHO. *Burning Opportunity: Clean Household Energy for Health, Sustainable Development, and Wellbeing of Women and Children*. World Health Organization, 2016. URL `http://www.who.int/airpollution/publications/burning-opportunities/en/`.

Evan Mills. Global Kerosene Subsidies: An Obstacle to Energy Efficiency and Development. *World Development*, 99:463–480, 2017.

William S Cleveland. Robust Locally Weighted Regression and Smoothing Scatterplots. *Journal of the American statistical association*, 74(368):829–836, 1979.

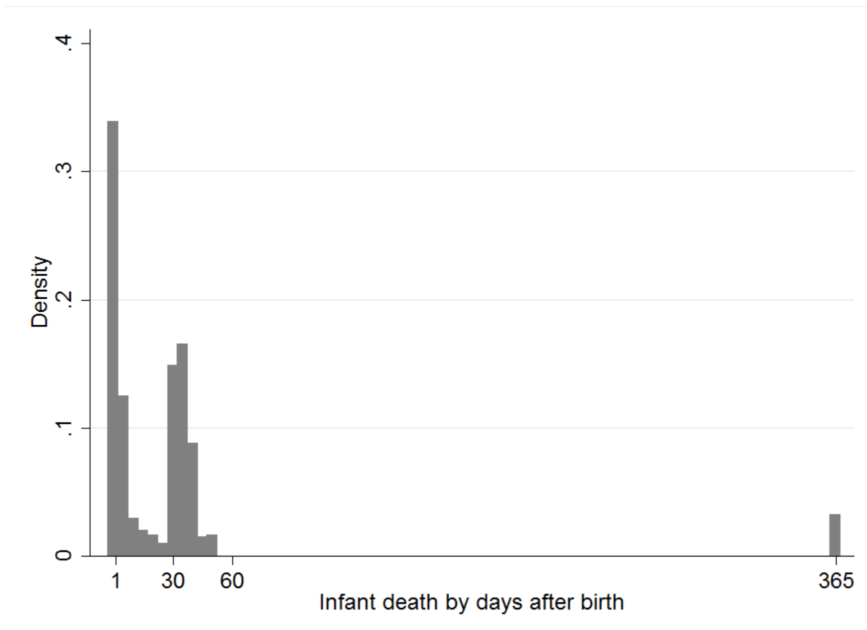
APPENDIX

Figure A.1: CHANGES IN MIGRATION RATES ACROSS DISTRICTS DURING 2000-2010



Notes: This figure shows the changes in the migration across districts from 2000 to 2005 and from 2005 to 2010. Mobility across districts in Indonesia is very low, less than 5% of from the total population, on average. Hence, it is difficult to find any compelling reason that the program is correlated with selective migration.
Source: Indonesia Census 2000, 2005 and 2010.

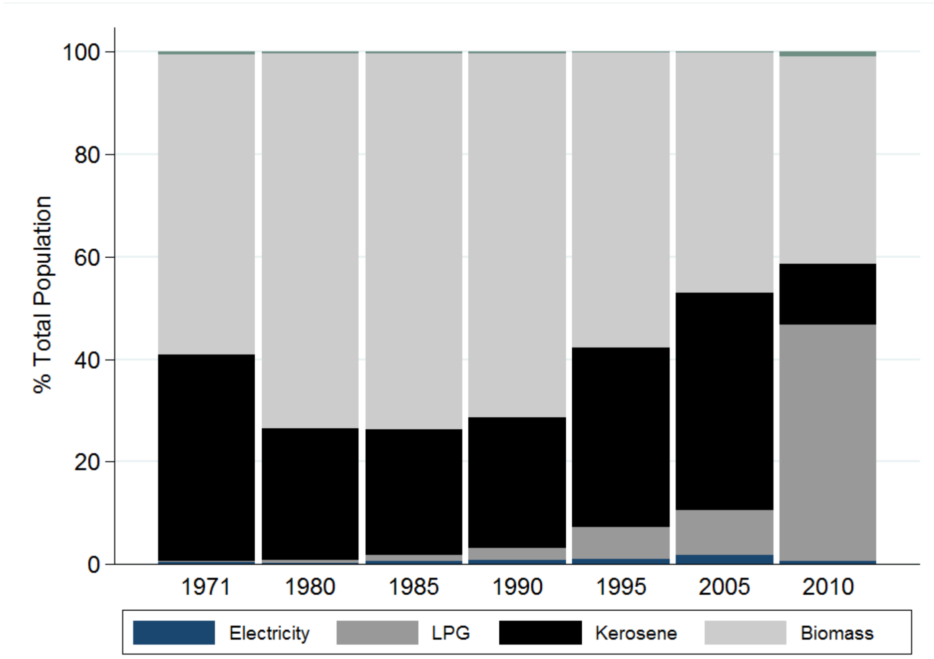
Figure A.2: HISTOGRAM OF INFANT MORTALITY BY DAYS AFTER BIRTH



Notes: This figure shows the histogram of infant mortality by days after birth with a third of those mortality occurred within the first day after birth.
Source: IDHS 2002-2012.

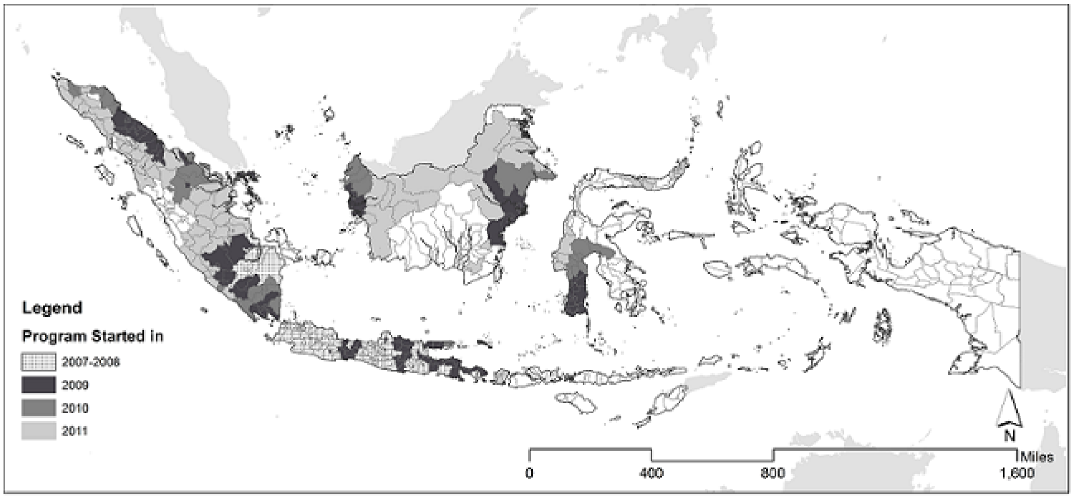
FIGURES

Figure 1: HOUSEHOLDS' PRIMARY COOKING FUEL DURING 1971-2010



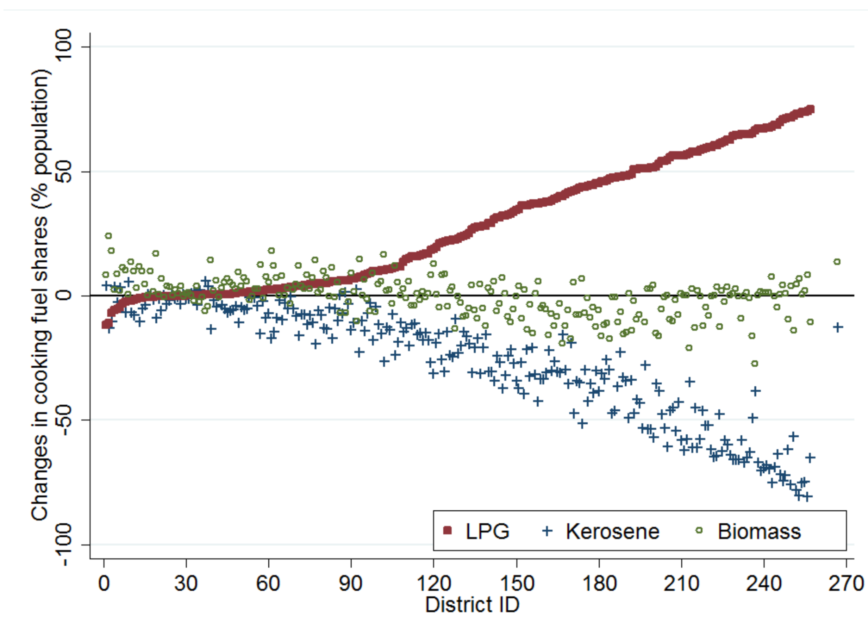
This figure shows the total population by primary cooking fuel types during 1971-2010. The share of households who used kerosene was stable prior to the program. However, after the program rolled out in 2007, the total population who use kerosene dropped from 42% to 12%, and the total population who used LPG increased from 9% to 46% within only three years.
Source: Indonesian census 1971-2010.

Figure 2: GEOGRAPHIC VARIATION OF THE PROGRAM



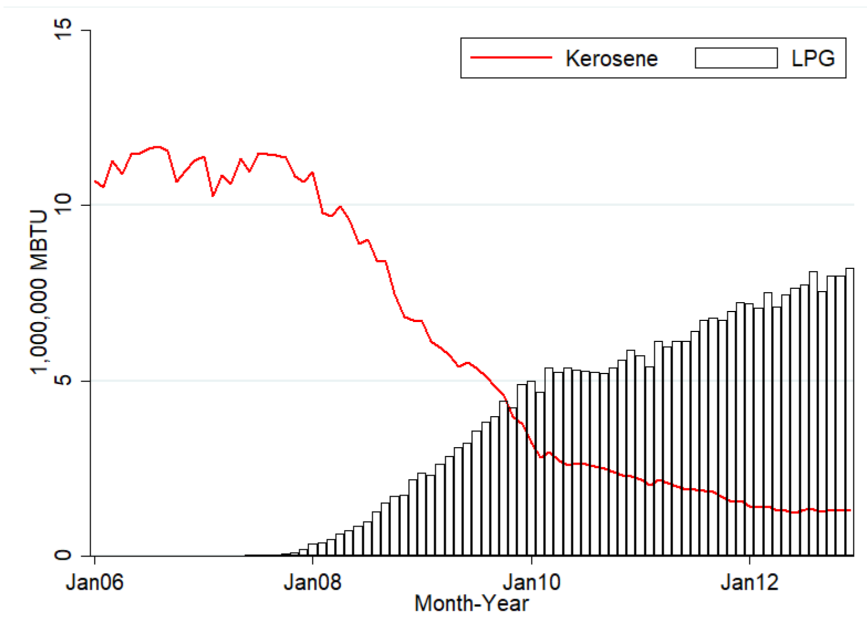
Notes: This figure plots the program implementation time within the study period. Big cities with large population such as Jakarta and Denpasar are among the early targeted districts, while the remaining districts that are less densely populated and farther from the ports are among the late targeted districts.
Source: Pertamina

Figure 3: CHANGES IN COOKING FUEL SHARES ACROSS DISTRICTS



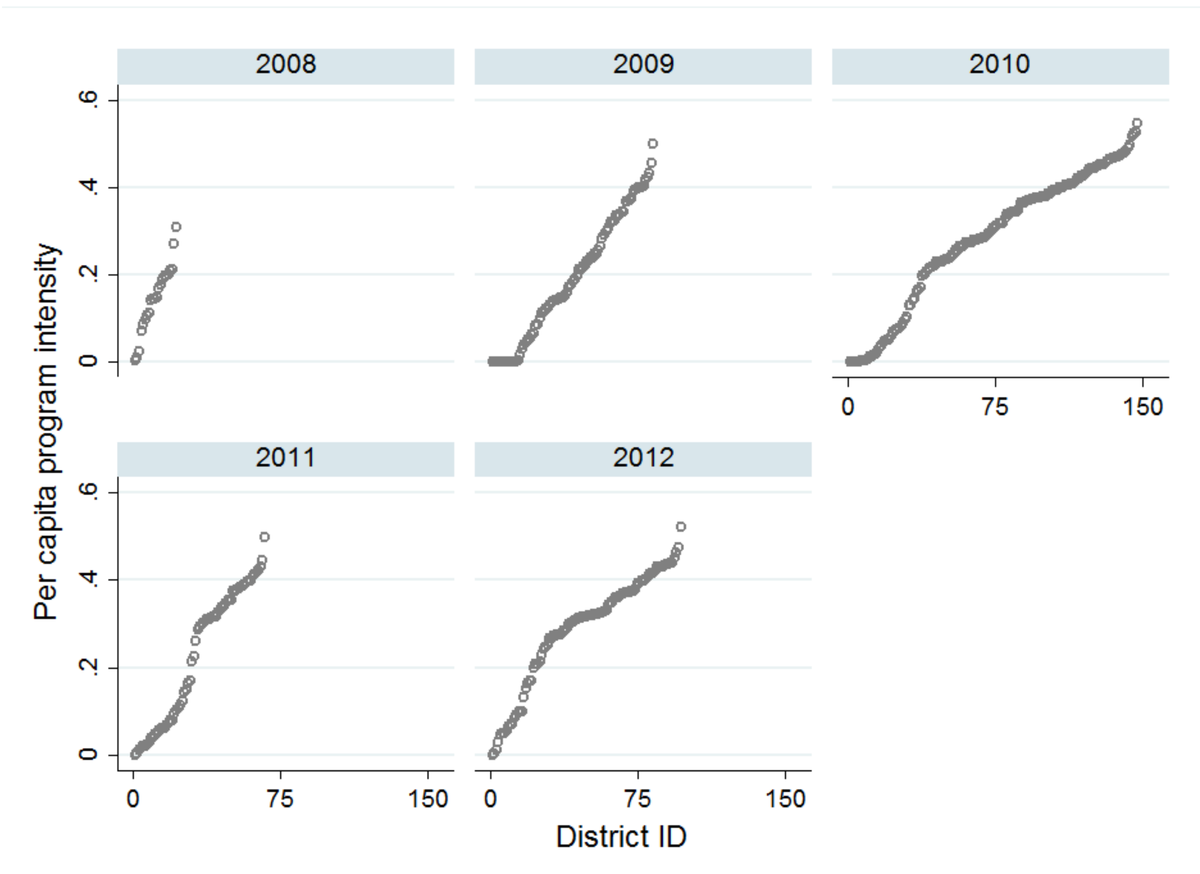
Notes: This figure shows the changes in district cooking fuel share (LPG, kerosene, and biomass) from 2005 to 2010. Each point indicates the total households that reported LPG or kerosene as their primary cooking fuel relative to the district's population (for the calculations, see Footnote 12). Districts are sorted by districts with the smallest change in the LPG shares.
Source: Indonesia Census 2005 and 2010.

Figure 4: CHANGES IN KEROSENE AND LPG CONSUMPTION OVER TIME



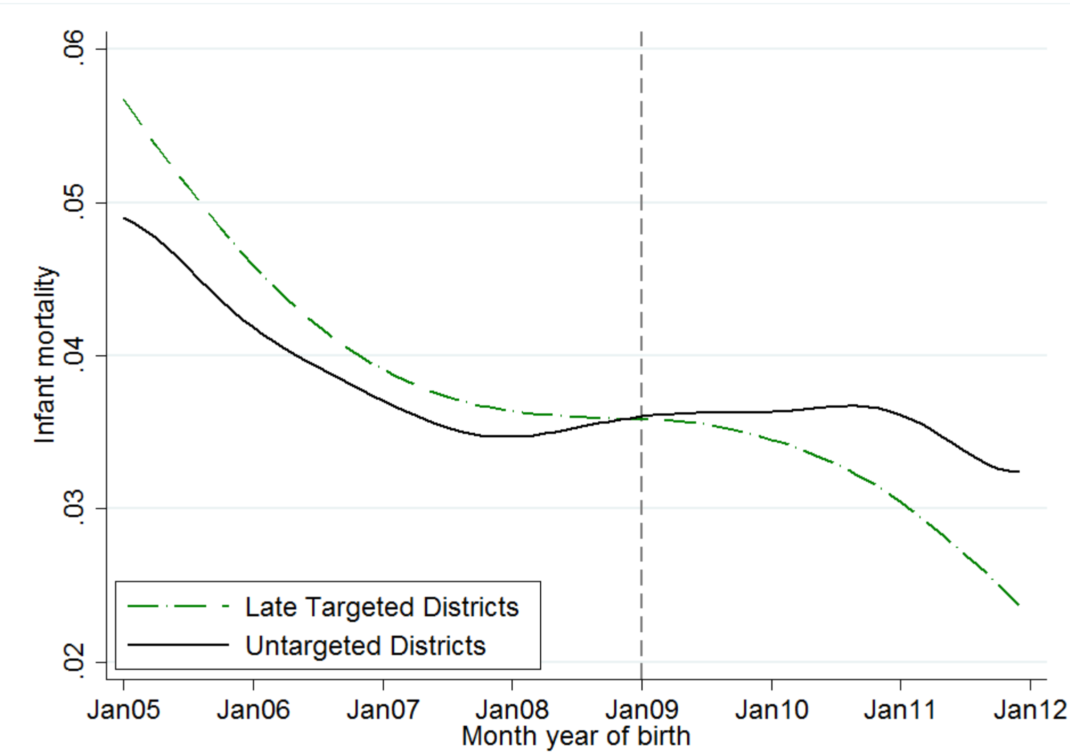
Notes: This figure shows the total quantity of kerosene and LPG supplied in each month-year, converted from the original unit to MBTU.
Source: Pertamina.

Figure 5: PROGRAM INTENSITY IN EACH DISTRICT BY YEAR



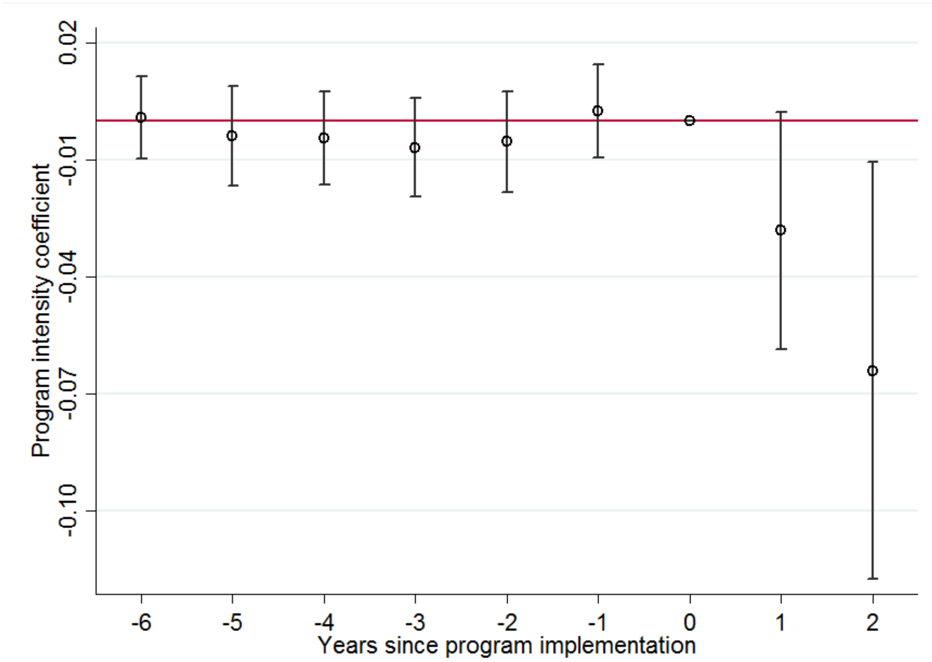
Notes: This figure shows the program intensity in each year in each district. In each year, district ID is sorted by the intensity level.
Source: Pertamina.

Figure 6: TRENDS IN INFANT MORTALITY



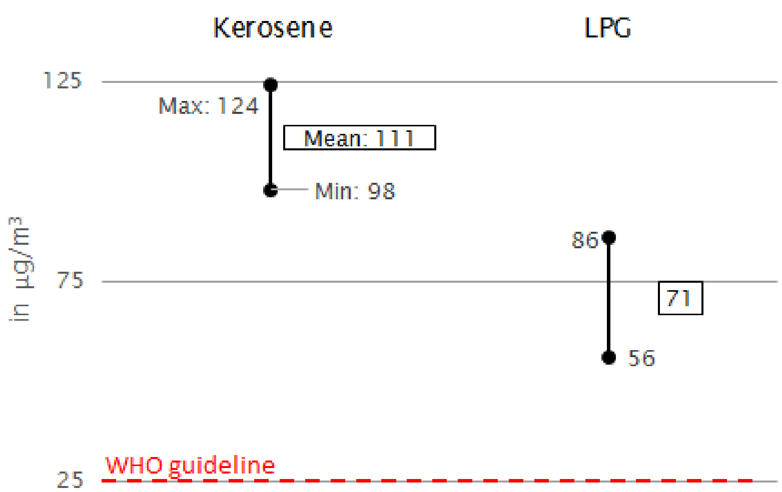
Notes: This figure plots the late targeted and untargeted districts using locally weighted regression with a tricube weighting function (Cleveland, 1979) and a bandwidth of 1. The vertical line indicates the beginning of the program roll-out. Note that each district had different program timing, due to the nature of the program roll-out.

Figure 7: EVENT STUDY PLOT



Notes: This figure plots the program intensity coefficient θ in Equation 2 by years prior to the program implementation. Each coefficient represents the mean difference between the treatment group and the control group. The year 0 since the program implementation is used as the reference category. Three years after the program implementation is excluded due to very few observations.

Figure 8: AVERAGE PERSONAL EXPOSURE OF $PM_{2.5}$ AMONG WOMEN WHO USE KEROSENE AND LPG



Notes: The figure illustrates the results from Andresen et al. (2005) which reflects the arithmetic mean and standard error of personal exposure during summertime June/July 2002 in India. The dashed vertical line shows WHO guideline for average $PM_{2.5}$ 24-hour mean concentration, $25\,\mu g\,m^{-3}$.

TABLES

Table 1: SUMMARY STATISTICS OF BASELINE CHARACTERISTICS

Variable	Treatment group: All targeted	Treatment group: Early targeted (2007-2008)	Treatment group: Late targeted (2009-2011)	Control group
	(1)	(2)	(3)	(4)
Infant death	0.034	0.028	0.037	0.040
	(0.181)	(0.166)	(0.190)	(0.196)
Mortality within 1 day	0.010	0.008	0.013	0.012
	(0.101)	(0.088)	(0.112)	(0.107)
Cooking with LPG	0.10	0.14	0.08	0.04
	(0.30)	(0.35)	(0.27)	(0.21)
Cooking with kerosene	0.40	0.49	0.37	0.25
	(0.49)	(0.50)	(0.48)	(0.43)
Cooking with wood	0.47	0.35	0.51	0.67
	(0.50)	(0.48)	(0.50)	(0.47)
First birth	0.35	0.38	0.34	0.29
	(0.48)	(0.48)	(0.47)	(0.45)
Antenatal visits	7.17	8.16	6.53	5.86
	(3.72)	(3.62)	(3.58)	(3.58)
Age at birth	27.57	27.64	27.44	27.69
	(6.32)	(6.30)	(6.29)	(6.45)
Mother's age <19	0.05	0.05	0.06	0.05
	(0.22)	(0.21)	(0.23)	(0.21)
First birth	0.35	0.38	0.34	0.29
	(0.48)	(0.48)	(0.47)	(0.45)
Children born in the last 5 years	1.32	1.24	1.35	1.47
	(0.54)	(0.47)	(0.56)	(0.63)
Number of household members	5.52	5.40	5.48	5.93
	(2.13)	(2.08)	(2.05)	(2.40)
Has TV	0.67	0.79	0.62	0.46
	(0.47)	(0.41)	(0.49)	(0.50)
Has fridge	0.23	0.29	0.19	0.16
	(0.42)	(0.45)	(0.40)	(0.37)
Has clean water for drinking	0.28	0.33	0.26	0.19
	(0.45)	(0.47)	(0.44)	(0.39)
Visited health facility last 12 months	0.49	0.51	0.48	0.49
	(0.50)	(0.50)	(0.50)	(0.50)
Not smoking	0.99	0.98	0.99	0.98
	(0.12)	(0.13)	(0.11)	(0.13)
House without toilet	0.56	0.45	0.61	0.71
	(0.50)	(0.50)	(0.49)	(0.45)
Has electricity	1.03	1.08	1.05	0.86
	(0.97)	(0.83)	(1.07)	(1.04)
Mother is not educated	0.04	0.02	0.04	0.07
	(0.19)	(0.15)	(0.20)	(0.26)
Father is not educated	0.03	0.02	0.04	0.05
	(0.17)	(0.12)	(0.20)	(0.22)
Mother's education: secondary and higher	0.53	0.56	0.53	0.47
	(0.50)	(0.50)	(0.50)	(0.50)
Father's education: secondary and higher	0.57	0.58	0.57	0.52
	(0.50)	(0.49)	(0.50)	(0.50)
Observations	28,682	6,405	12,446	9,831

Notes: This table reports the average households' characteristics at baseline in the targeted districts (column 1), targeted districts during 2007-2008 (Column 2), targeted districts during 2009-2011 (Column 3), and untargeted districts (Column 4). All numbers are weighted using DHS sample weights. Standard deviations are in parentheses.
Source: IDHS 2002 and 2007.

Table 2: BALANCING CHECKS ON BASELINE CHARACTERISTICS

	Treatment: all		Treatment: 2007-2008		Treatment: 2009-2011	
	Coef.	SE	Coef.	SE	Coef.	SE
	(1)	(2)	(3)	(4)	(5)	(6)
Infant death	-0.007	(0.006)	-0.004	(0.007)	-0.010	(0.007)
Infant death within 1 day	0.001	(0.003)	-0.000	(0.003)	0.001	(0.003)
Cooking with LPG	0.018	(0.015)	0.022	(0.023)	0.023	(0.014)
Cooking with kerosene	-0.032	(0.033)	-0.043	(0.034)	-0.021	(0.037)
Cooking with wood	0.006	(0.037)	0.005	(0.038)	-0.006	(0.041)
First birth	0.010	(0.013)	0.012	(0.016)	0.013	(0.014)
Antenatal visits	-0.218	(0.262)	-0.028	(0.284)	-0.213	(0.267)
Age at birth	-0.147	(0.240)	-0.083	(0.282)	-0.156	(0.253)
Mother's age <19	-0.003	(0.007)	-0.009	(0.009)	-0.000	(0.008)
First birth	0.010	(0.013)	0.012	(0.016)	0.013	(0.014)
Children born in the last 5 years	-0.049	(0.031)	-0.057*	(0.032)	-0.052	(0.034)
Number of household members	-0.167	(0.146)	-0.200	(0.163)	-0.149	(0.151)
Has TV	-0.051	(0.034)	-0.058*	(0.032)	-0.034	(0.038)
Has fridge	0.018	(0.020)	0.020	(0.027)	0.027	(0.021)
Has clean water for drinking	0.062**	(0.030)	0.125***	(0.037)	0.038	(0.033)
Visited health facility last 12 months	0.009	(0.038)	0.012	(0.042)	0.013	(0.040)
Not smoking	0.004	(0.004)	-0.002	(0.005)	0.008**	(0.004)
House without toilet	0.061**	(0.030)	0.083**	(0.034)	0.037	(0.033)
Has electricity	0.055	(0.046)	0.046	(0.053)	0.064	(0.050)
Mother is not educated	-0.031**	(0.013)	-0.029**	(0.014)	-0.034**	(0.014)
Father is not educated	-0.015	(0.009)	-0.013	(0.009)	-0.017*	(0.010)
Mother's education: secondary and higher	0.001	(0.029)	0.008	(0.033)	0.001	(0.032)
Father's education: secondary and higher	-0.044	(0.027)	-0.031	(0.030)	-0.046	(0.030)
Observations	28,682		16,236		22,277	

Notes: Each row and column is a separate regression that includes the simplest model with year and district fixed effects on the corresponding dependent variable in each row. All regressions use the untargeted districts as the control group. In the treatment group, column 1 uses all targeted districts, column 3 uses early targeted districts, and column 5 uses late targeted districts. Columns 1, 3, and 5 report the regression coefficient for the treatment indicator, while columns 2, 4, and 6 report its corresponding standard errors. Standard errors (in parentheses) are clustered by district. *** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.
Source: IDHS 2002 and 2007.

Table 3: CORRELATION BETWEEN THE OBSERVABLES AND THE TREATMENT VARIABLE

	Program dummy				Program Intensity			
Treatment group:	All targeted		Targeted in 2009-2011		All targeted		Targeted in 2009-2011	
Dependent Variable	Coef.	SE	Coef.	SE	Coef.	SE	Coef.	SE
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Cooking with LPG	0.234***	(0.016)	0.205***	(0.017)	0.352***	(0.046)	0.395***	(0.054)
Cooking with kerosene	-0.240***	(0.019)	-0.184***	(0.018)	-0.306***	(0.037)	-0.295***	(0.042)
Cooking with wood	0.008	(0.013)	-0.020	(0.016)	-0.022	(0.037)	-0.065	(0.053)
Number of household members	-0.061	(0.069)	0.038	(0.067)	-0.032	(0.152)	0.012	(0.187)
Has TV	-0.040***	(0.013)	0.005	(0.014)	0.028	(0.031)	0.082**	(0.040)
Has fridge	-0.009	(0.014)	0.009	(0.015)	0.004	(0.037)	0.036	(0.049)
Has clean water for drinking	0.022	(0.016)	0.015	(0.019)	-0.031	(0.042)	-0.060	(0.054)
Visited health facility last 12 months	0.017	(0.017)	0.021	(0.018)	0.009	(0.043)	-0.022	(0.056)
Not smoking	0.007*	(0.004)	0.008*	(0.004)	-0.003	(0.011)	0.005	(0.013)
House without toilet	0.019	(0.014)	-0.005	(0.017)	-0.013	(0.039)	-0.039	(0.046)
Has electricity	-0.080***	(0.023)	-0.054*	(0.031)	-0.204***	(0.070)	-0.256***	(0.097)
Mother's education: secondary and higher	-0.004	(0.012)	-0.000	(0.015)	0.038	(0.040)	0.108**	(0.047)
Father's education: secondary and higher	-0.002	(0.012)	0.001	(0.014)	0.016	(0.038)	0.043	(0.045)
Antenatal visits	-0.081	(0.129)	0.182	(0.124)	-0.233	(0.305)	0.013	(0.410)
Age at birth	-0.045	(0.138)	-0.172	(0.182)	0.498	(0.531)	0.110	(0.616)
Mother's age <19	0.002	(0.005)	0.006	(0.006)	-0.008	(0.016)	-0.003	(0.021)
First birth	-0.003	(0.011)	-0.002	(0.014)	-0.011	(0.044)	0.004	(0.052)
Children born in the last 5 years	0.035***	(0.013)	0.016	(0.015)	0.038	(0.039)	0.018	(0.053)
Observations	46,750		36,443		46,307		36,097	

Notes: Each row and column is a separate regression that includes the simplest model with year and district fixed effects as the set of control variables with the corresponding dependent variable in each row. Columns 1-4 use program dummy and columns 5-8 use program intensity as the treatment variable. Columns 1-2 and columns 5-6 use all sample while columns 3-4 and columns 7-8 use only late targeted regions. Columns 1, 3, 5, and 7 report the regression coefficient for treatment indicator if households are in the targeted districts, while columns 2, 4, 6, and 8 report its corresponding standard errors. Antenatal visits variable is recorded for the latest birth and has a smaller sample compared to the rest. Standard errors (in parentheses) are clustered by district. *** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.
Source: IDHS 2002, 2007, 2014

Table 4: THE PROGRAM IMPACT ON INFANT MORTALITY USING DUMMY PROGRAM

	Dummy program Mean of infant mortality: 37 per 1,000 live birth					
Variables	(1)	(2)	(3)	(4)	(5)	(6)
Program dummy	-0.019***	-0.017***	-0.011**	-0.010**	-0.011***	-0.012*
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.006)
Has TV				0.003	0.002	0.003
				(0.003)	(0.004)	(0.004)
Has fridge				-0.002	0.004	0.003
				(0.003)	(0.004)	(0.004)
Has clean water for drinking				-0.004*	-0.006*	-0.004
				(0.002)	(0.004)	(0.004)
Visited health facility last 12 months				-0.009***	-0.012***	-0.013***
				(0.002)	(0.003)	(0.003)
Not smoking				-0.027**	-0.024	-0.025
				(0.012)	(0.016)	(0.017)
House without toilet				0.005*	0.004	0.004
				(0.003)	(0.004)	(0.004)
Has electricity				-0.006	-0.006	-0.008
				(0.005)	(0.005)	(0.005)
Mother's education: secondary and higher				-0.010***	-0.009**	-0.009**
				(0.003)	(0.003)	(0.004)
Father's education: secondary and higher				0.000	-0.000	0.000
				(0.003)	(0.003)	(0.004)
Mother is not educated				-0.001	-0.004	-0.002
				(0.008)	(0.008)	(0.009)
Father is not educated				0.012	0.023**	0.024**
				(0.009)	(0.010)	(0.010)
Age at birth				0.000	0.223	0.181
				(0.000)	(0.391)	(0.382)
Mother's age <19				0.017***	7.783	6.299
				(0.006)	(14.149)	(13.852)
Birth order (2nd)				-0.003	0.002	0.002
				(0.003)	(0.004)	(0.004)
Birth order (3rd)				0.002	0.010**	0.009*
				(0.003)	(0.005)	(0.005)
Birth order (4th)				0.007	0.014**	0.014**
				(0.005)	(0.006)	(0.006)
Observations	35,092	35,092	35,092	35,092	35,092	35,092
R-squared	0.002	0.035	0.052	0.055	0.059	0.083
Year fixed effects	Y	Y	Y	Y	Y	Y
Birth characteristics		Y	Y	Y	Y	Y
District fixed effects			Y	Y	Y	Y
Full control variables				Y	Y	Y
Controls X post program					Y	Y
District trends						Y

Notes: The dependent variable is infant mortality which takes value 1 if the infant is dead and 0 otherwise. In a cumulative fashion, column 1 uses year fixed effects, column 2 adds district fixed effects, column 3 adds households and individual characteristics, column 4 adds district-specific linear trends, column 5 adds district-specific quadratic trends, and column 6 adds all interactions between the control variables and the post-program dummy. Standard errors (in parentheses) are clustered by district. *** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.

Table 5: THE PROGRAM IMPACT ON INFANT MORTALITY USING PROGRAM INTENSITY

	Program intensity Mean of infant mortality: 37 per 1,000 live birth					
Variables	(1)	(2)	(3)	(4)	(5)	(6)
Program intensity	-0.046***	-0.041***	-0.033**	-0.032**	-0.033**	-0.021
	(0.014)	(0.014)	(0.014)	(0.015)	(0.015)	(0.018)
Has TV				0.003	0.002	0.003
				(0.003)	(0.004)	(0.004)
Has fridge				-0.001	0.004	0.003
				(0.003)	(0.004)	(0.004)
Has clean water for drinking				-0.004	-0.006*	-0.004
				(0.002)	(0.004)	(0.004)
Visited health facility last 12 months				-0.009***	-0.012***	-0.013***
				(0.002)	(0.003)	(0.003)
Not smoking				-0.028**	-0.024	-0.025
				(0.012)	(0.016)	(0.017)
House without toilet				0.005*	0.004	0.004
				(0.003)	(0.004)	(0.004)
Has electricity				-0.006	-0.006	-0.008
				(0.005)	(0.005)	(0.005)
Mother's education: secondary and higher				-0.010***	-0.009**	-0.009**
				(0.003)	(0.003)	(0.004)
Father's education: secondary and higher				-0.000	-0.000	0.000
				(0.003)	(0.003)	(0.004)
Mother is not educated				-0.003	-0.004	-0.002
				(0.008)	(0.008)	(0.009)
Father is not educated				0.012	0.023**	0.024**
				(0.009)	(0.010)	(0.010)
Age at birth				0.000	0.223	0.181
				(0.000)	(0.391)	(0.382)
Mother's age <19				0.018***	7.768	6.300
				(0.006)	(14.156)	(13.856)
Birth order (2nd)				-0.003	0.002	0.002
				(0.003)	(0.004)	(0.004)
Birth order (3rd)				0.002	0.010**	0.009*
				(0.003)	(0.005)	(0.005)
Birth order (4th)				0.007	0.014**	0.014**
				(0.005)	(0.006)	(0.006)
Observations	34,755	34,755	34,755	34,755	34,755	34,755
R-squared	0.001	0.035	0.052	0.056	0.060	0.083
Year fixed effects	Y	Y	Y	Y	Y	Y
Birth characteristics		Y	Y	Y	Y	Y
District fixed effects			Y	Y	Y	Y
Full control variables				Y	Y	Y
Controls X post program					Y	Y
District trends						Y

Notes: The dependent variable is infant mortality which takes value 1 if the infant is dead and 0 otherwise. In a cumulative fashion, column 1 uses year fixed effects, column 2 adds district fixed effects, column 3 adds households and individual characteristics, column 4 adds district-specific linear trends, column 5 adds district-specific quadratic trends, and column 6 adds all interactions between the control variables and the post-program dummy. Standard errors (in parentheses) are clustered by district. *** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.

Table 6: THE PROGRAM IMPACTS ON INFANT MORTALITY WITH ALTERNATIVE SPECIFICATIONS

	Within rural areas					Within mother
Variables	(1)	(2)	(3)	(4)	(5)	(6)
Panel 1: Program impacts using dummy program						
Dummy program	-0.016***	-0.011**	-0.007	-0.005	-0.004	-0.011
	(0.005)	(0.005)	(0.005)	(0.005)	(0.007)	(0.018)
Observations	26,424	26,424	26,424	26,424	26,424	11,921
R-squared	0.001	0.038	0.060	0.064	0.101	0.546
Panel 2: Program impacts using program intensity						
Program Intensity	-0.068***	-0.050***	-0.042***	-0.040***	-0.034**	-0.035
	(0.014)	(0.014)	(0.014)	(0.014)	(0.017)	(0.073)
Observations	26,190	26,190	26,190	26,190	26,190	11,764
R-squared	0.001	0.038	0.061	0.065	0.101	0.547
Year fixed effects	Y	Y	Y	Y	Y	Y
Birth characteristics		Y	Y	Y	Y	Y
District fixed effects			Y	Y	Y	
Full control variables				Y	Y	
Controls X post program					Y	
District trends					Y	
Mother fixed effects						Y

Notes: The dependent variable is infant mortality. Panel 1 shows the program impacts using program dummy (similar to Table 4) and Panel 2 shows the program impacts using program intensity (similar to Table 5). Columns 1-5 use only samples in rural areas, and column 6 uses only the sample of all mothers with more than one birth in the late targeted district. Standard errors (in parentheses) are clustered by district. *** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.

Table 7: THE PROGRAM EFFECT ON BIRTH OUTCOMES

Variables	(1) within 1 day	(2) Deaths at age 2-30 days	(3) 1-11 months	(4) Low birth weight	(5) Very low birth weight
Program Intensity	-0.020**	0.011	-0.005	-0.041	-0.018*
	(0.008)	(0.011)	(0.009)	(0.037)	(0.011)
Observations	34,757	34,757	34,757	25,722	25,722
R-squared	0.060	0.052	0.057	0.114	0.097
Mean dep. var	11.7	9.2	15.2	134.9	8.6
Year fixed effects	Y	Y	Y	Y	Y
Birth characteristics	Y	Y	Y	Y	Y
District fixed effects	Y	Y	Y	Y	Y
Full control variables	Y	Y	Y	Y	Y
Controls X post program	Y	Y	Y	Y	Y
District trends	Y	Y	Y	Y	Y

Notes: Each column is a separate regression of program intensity on an outcome variable indicated in the column header. All regressions include year and district fixed effects, district-specific linear and quadratic trend, full control variables discussed in Section III.C and all of the interactions between the control variables and the post-program dummy. The mean of each outcome variables is measured at baseline and per 1,000 live births. Standard errors (in parentheses) are clustered by district. *** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.

Table 8: PROGRAM EFFECT AMONG KEROSENE USERS AND ELECTRIFIED HOUSEHOLDS

Variables	(1) Kerosene is the primary cooking fuel	(2) Kerosene is not the primary cooking fuel	(3) Have access to electricity	(4) Have no access to electricty
Program intensity	-0.018	-0.034**	-0.034**	0.082
	(0.053)	(0.016)	(0.015)	(0.101)
Observations	9,950	24,805	28,728	6,027
R-squared	0.084	0.057	0.054	0.103

Notes: Each column is a different regression with different subsamples. Column 1 uses only households who cook with kerosene, column 2 uses only households who do not use kerosene, column 3 uses only households who have access to electricity, and column 4 uses only households who do not have access to electricity. The dependent variable is infant mortality. All the regressions use a full set of controls: year fixed effects, district fixed effects, district-specific linear trend, district-specific quadratic trend, control variables discussed in Section III.C and its interaction with the post-program dummy. Standard errors (in parentheses) are clustered by district. *** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level.