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## Association between wood cooking fuel and maternal hypertension at delivery in central East India

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### Abstract

**Objective**—Smoke from burning of biomass fuels has been linked with adverse pregnancy outcomes and with hypertension among nonpregnant subjects; association with hypertension during pregnancy has not been well studied. We sought to evaluate whether use of wood cooking

fuel increases the risk of maternal hypertension at delivery compared to gas which burns with less smoke.

**Methods**—Information on fuel use and blood pressure was available for analysis from a cross-sectional survey of 1369 pregnant women recruited at delivery in India.

**Results**—Compared to gas users, women using wood as fuel had on average lower mean arterial pressure (adjusted effect size  $-2.0$  mmHg; 95% CI  $-3.77, -0.31$ ) and diastolic blood pressure (adjusted effect size  $-1.96$  mmHg; 95% CI  $-3.60, -0.30$ ) at delivery. Risk of hypertension (systolic  $> 139$  mmHg or diastolic  $> 89$  mmHg) was 14.6% for women cooking with wood compared to 19.6% for those cooking with gas although this did not reach significance after adjustment, using propensity score techniques, for factors that make wood and gas users distinct (adjusted prevalence ratio 0.76; 95% CI 0.49, 1.17).

**Conclusions**—Combustion products from the burning of biomass fuels are similar to those released with tobacco smoking which has been linked with a reduced risk for preeclampsia. The direction of our findings suggests the possibility of a similar effect for biomass cook smoke. Whether clean cook cooking interventions being promoted by international advocacy organizations will impact hypertension in pregnancy warrants further analysis as hypertension remains a leading cause of maternal death worldwide and cooking with biomass fuels is widespread.

### Keywords

biomass cook smoke; pregnancy; hypertension; gestational hypertension; household air pollution

## INTRODUCTION

Biomass fuels such as wood, charcoal, and crop residues are the primary energy source for cooking and/or heating for an estimated 2.8 billion people, almost half of the world's population.<sup>1</sup> Air pollution generated from the inefficient combustion of these solid fuels has been recognized as a major contributor to the global burden of disease. Recent estimates suggest that smoke generated from biomass burning accounts for over 4 million premature deaths among children and adults from pneumonia, lung cancer, chronic lung disease or cardiovascular disease.<sup>2,3</sup> More than a quarter (27%) of the people using solid fuels worldwide resides in India.<sup>4</sup>

For women and girls globally, household air pollution ranks as the second most important risk factor contributing to disability-adjusted life years lost.<sup>2</sup> As women of reproductive age are the primary cooks in many households, there has been increasing attention devoted to the potential adverse effects of household air pollution on pregnancy outcomes. Observational studies suggest that biomass smoke exposure during pregnancy may decrease birth weight and increase the risk of low birth weight and stillbirth.<sup>5-12</sup> We have also reported an increased risk of preterm birth among Indian women cooking with wood during pregnancy compared to those cooking with gas.<sup>13</sup>

Despite a growing focus on birth outcomes following exposure to household air pollution, little attention has been devoted to maternal outcomes. A family of pregnancy complications

that might plausibly be linked with cooking smoke is hypertensive disorders of pregnancy which includes preeclampsia/eclampsia, gestational hypertension, and chronic hypertension with or without superimposed preeclampsia. Household air pollution from biomass burning has been linked with both increases in blood pressure<sup>14,15</sup> and chronic hypertension<sup>16–18</sup> among nonpregnant populations in several observational cohorts. Introduction of an improved cook stove reduced diastolic blood pressure among Guatemalan cooks<sup>20</sup> and reduced systolic blood pressure among older cooks in a recent Nicaraguan trial.<sup>21</sup> No studies have evaluated the risk of gestational hypertension or preeclampsia/eclampsia among women cooking with biomass fuels although outdoor air pollution has been associated with an increased risk of gestational hypertension in most<sup>22–28</sup> but not all studies.<sup>29,30</sup> This is in contrast to the well documented decreased risk for preeclampsia among cigarette smokers.<sup>31</sup> Since hypertension is a major cause of maternal death<sup>32</sup>, it is critical to determine whether pollution generated from the burning of biomass fuels affects this risk. Policymakers will want to know whether efforts to increase the number of households cooking with clean energy might translate into a reduction in maternal deaths from hypertension.

We used data from two similarly conducted cross sectional surveys of pregnant women enrolled at delivery in India to evaluate the relationship between hypertension in pregnancy and wood cooking fuel, as a marker of household air pollution from biomass burning. Due to the fairly rudimentary delivery settings, information about proteinuria and other end organ involvement was not available; consequently, we were limited in our ability to distinguish between the various hypertensive disorders of pregnancy. Measured hypertension in our cohort therefore represents a mix of preeclampsia/eclampsia, gestational hypertension, and underlying chronic hypertension.

We recognize that women cooking with wood are likely to be systematically poorer than women who cook with gas. An identified association between cooking fuel and hypertension could therefore be biased by these socioeconomic factors as has been suggested in our previous work with regards to low birth weight and other birth outcomes.<sup>13</sup> We employed propensity score techniques to address this concern and to increase the statistical power to simultaneously adjust for multiple confounders.<sup>33</sup>

## METHODS

### Study Sites/Procedures

Information regarding self-reported primary cooking fuel, time spent cooking and maternal blood pressure measurements was available for secondary analysis from two cross-sectional studies of pregnant women in central east India that were conducted to establish the burden of malaria during pregnancy.<sup>34,35</sup> In these two studies, all women aged 15 years or older who presented for delivery at the study sites were approached for consent. Enrollment occurred over twelve months in Jharkhand state beginning in December 2006 at one urban and two rural facilities. In neighboring Chhattisgarh, recruitment occurred from June 2007 to May 2008 in two urban and two rural facilities. As case report forms and study procedures were the same in both cohorts, data were concatenated from the two sites to increase power and generalizability of this secondary analysis.

Following consent, women were interviewed about a variety of demographic, economic, obstetric and medical factors. Information obtained included the primary cooking fuel used in their household and average number of hours per day they spent cooking during their pregnancy. A physical examination was performed which included measurement of blood pressure, height and weight. For blood pressure, trained research staff followed a standardized protocol using an appropriately sized cuff with the subject's arm supported at the level of the heart. The systolic blood pressure (SBP) was recorded as the mmHg visible on the mercury manometer when the pulse was first auscultated. The diastolic blood pressure (DBP) was recorded when the auscultated pulse disappeared. The blood pressure measurements were repeated twice per subject by the same research staff and averaged to represent the SBP and DBP. The history and physical examination occurred at a convenient time after admission to the ward and before 24 hours had lapsed since delivery. The maternal physical examination occurred after delivery for over 87% of the subjects. We therefore limited analyses to women with postpartum measurements of blood pressure to represent a more uniform sample as intrapartum measurements may be elevated during contractions or from pain. Vital signs during the course of the labor and delivery were neither consistently obtained nor recorded by the managing clinical team. Blood pressure measurements were consequently limited to those obtained by research staff. Further details of the original studies less relevant to this analysis are presented elsewhere.<sup>34–37</sup>

### Definitions

A subject's body mass index (BMI) was calculated by dividing her weight in kilograms at the time of the physical examination by her height in meters squared:  $BMI = \text{weight (in kilograms)} / \text{height (in meters)}^2$ . Hypertension at delivery (HTN) was defined as a SBP of greater than 139 mmHg or a DBP of greater than 89 mmHg. Severe hypertension at delivery (severe HTN) was defined as a SBP of greater than 159 mmHg or a DBP of greater than 109 mmHg. Information about maternal proteinuria was not uniformly available in the cohort; this was consequently not used in our definition of hypertension at delivery. Mean arterial pressure (MAP) was calculated using the SBP and DBP measurements as follows:  $MAP = (2/3 * DBP) + (1/3 * SBP)$ . Nulliparity was defined as having delivered no prior infant after the onset of fetal quickening, whether or not the infant was born alive or stillborn.

### Data Analysis

We restricted our analyses to a comparison of women cooking with wood to those cooking with gas rather than including a broader range of potentially non-comparable fuel types<sup>38–40</sup> (charcoal, cow dung, kerosene) as wood and gas were used by more than 90% of women in our cohort.

**Propensity- score model**—To address the systematic differences between women cooking with wood versus gas, a propensity score model was fit. Women who primarily cooked with wood were compared to women cooking with gas across a number of variables that were potentially linked with exposure (wood cooking fuel) but which might be confounded by poverty. Odds ratios and c-statistics were calculated to explore the association of each covariate with wood fuel use. The final variables for the propensity score model were chosen based on the strength of the association and the prevalence of the

predictor. A propensity score was then calculated for each subject using this model which represents the predicated probability that the subject's primary household fuel was wood (range 0 to 1). We excluded from the propensity score modeling exercise obstetric or medical covariates that might be more strongly linked with hypertensive outcomes and instead considered these variables for inclusion separately in the final adjusted models. Exploration of the relationship between HTN and the propensity score suggested that the propensity score should be modeled nonlinearly. The propensity score was categorized into quintiles for multivariable modeling and can loosely be interpreted as a proxy for poverty.

**Unadjusted association between fuel use and HTN**—The proportions of subjects with HTN and severe HTN as well as the mean SBP, DBP, and MAP were compared between the two groups (users of wood versus gas). Categorical data are presented as frequency counts (percent) and compared using the Pearson chi-square or Fisher's exact as appropriate. Continuous data are summarized as means ( $\pm$  standard deviation) and compared using analysis of variance. Unconditional log-binomial regression models were constructed to estimate univariable prevalence ratios and associated 95% confidence intervals (CIs) for HTN. The referent group was women cooking primarily with gas. Exact logistic regression was used to estimate an odds ratio for severe HTN given the rarity of this outcome. Linear regression models were used to estimate the effect of wood fuel exposure on mean DBP, SBP, and MAP; 95% CIs were constructed using the modeled standard error.

**Adjusted association between fuel use and HTN**—Variables that might be plausibly linked with HTN were considered for inclusion in adjusted models by evaluating their univariate association with HTN. These covariates included maternal age, body mass index, nulliparity, multiple gestations, history of hypertension, history of diabetes mellitus, adequate antenatal clinic attendance (4 or more visits), maternal smoking, use of smokeless tobacco, and parasitemia. We also considered the presence of windows and the time spent cooking for potential inclusion as these might reflect exposure to cooking smoke. While the range of responses varied from 1 to 7 hours, almost all of the variability was limited to the upper decile of cooking time (4 or more hours of cooking). We therefore dichotomized time spent cooking into 4 or more hours versus less than 4 hours.

Multivariable log-binomial regression modeling was used to adjust the association of wood fuel use with HTN for the categorized propensity score, and variables we identified to be associated with HTN at a significance level of 0.05 or less. A final adjusted prevalence ratio and 95% CI was calculated based on these findings. The effect of fuel use on SBP, DBP and MAP was adjusted in multivariable linear regression using the same covariates chosen in the final adjusted HTN model. No adjustments were made to the severe HTN model given the rarity of this outcome. Data were missing for fewer than 5 subjects for the covariates included in the final model and therefore no additional methods were employed to handle missing data.

Statistical analyses were performed using SAS software version 9.2 (Cary, North Carolina).

## Ethical Clearance

The study was approved by the Boston University and Centers for Disease Control and Prevention Institutional Review Boards, the Ethics committee and the Scientific Advisory Committee of the National Institute of Malaria Research in India, and the Health Ministry Screening Committee of Indian Council of Medical Research.

## RESULTS

In the state of Jharkhand, a total of 739 pregnant women were screened at the time of delivery and all were eligible, although 21 declined participation. In the state of Chhattisgarh, all 1028 pregnant women screened were eligible; two women declined participation leaving a combined total of 1744 subjects from the two cohorts. For this analysis, we excluded two women for lack of blood pressure measurements, 174 women that did not use wood or gas as their primary fuel, and an additional 199 women that had only intrapartum blood pressure measurements recorded. A total of 1369 subjects remained, 1134 who cooked primarily with wood and 235 who cooked primarily with gas. Our decision to limit analysis to postpartum measurements of blood pressure was affirmed by the finding that the diagnosis of HTN was remarkably high among women with intrapartum measurements (66 of 195 subjects, 33.9%) compared with those who had postpartum measurements (212 of 1369, 15.5%;  $p<0.0001$ ).

### Comparison of wood and gas users and propensity score model

As anticipated, women who primarily cooked with wood were quite different than women cooking with gas (Table 1). They were less likely to be overweight, to have attended an adequate number of antenatal clinic visits, and to be taking iron and folate. With the exception of marital status, women cooking with wood differed across every socio-demographic characteristic we considered. They were more likely to be from a historically disadvantaged caste, to work in agricultural occupations, and to live in dwellings made with impermanent wall, roof, and floor materials. They were less likely to have completed more than 5 years of school, to own modern material comforts, or to have windows in their homes.

To address these systematic differences between women cooking with wood versus gas, a propensity score model was created. The model fit included 13 variables with a c-statistic of 0.951; the variables were impermanent roofing, impermanent walls, impermanent floors, caste, agricultural work, primary education, and ownership of a radio, electric fan, room cooler, television, refrigerator, motorcycle and four wheel vehicle. The mean propensity score among wood users was 0.93 and among gas users was 0.32 ( $p<0.0001$ ).

### Univariable association of wood fuel use with hypertension and blood pressure

Compared to women cooking with gas, 14.6% of women using wood as their primary fuel met criteria for HTN at delivery compared to 19.6% of gas users ( $p=0.0570$ ); prevalence ratio 0.75 [95% CI 0.56, 1.00] (Table 2). Mean DBP and MAP following delivery was significantly lower among women cooking with wood compared to those cooking with gas ( $p=0.001$  and  $p=0.0072$  respectively, Table 2). The effect size for both DBP and MAP was

less than 5 mmHg. There was no difference in the frequency of severe HTN or in mean SBP between the two groups.

### **Adjusted association of wood fuel use with hypertension and blood pressure**

Each covariate in Table 1 not included in the propensity model was evaluated for its univariate association with HTN at delivery. Only four variables were significantly related (Table 3); these covariates were cohort (Jharkhand versus Chhattisgarh), history of hypertension, presence of windows, and use of smokeless tobacco. While the proportion of women whose average daily cooking time was in the upper decile was much higher among wood users compared with gas users (17.8% vs. 8.9%,  $p=0.0008$ ), HTN was not more common among women who cooked longer (14.2% vs. 16.7%,  $p=0.36$ ). Other obstetric and maternal characteristics considered were not linked with HTN. Notably, there was no difference in the mean propensity score between hypertensive versus normotensive subjects (0.80 vs. 0.83,  $p=0.1028$ ) nor was there a linear relation between the frequency of HTN and quintiles of propensity score ( $p=0.1790$ ). Furthermore, of all the covariates included in the propensity score model, only TV ownership was associated with HTN at a significance level of  $p < 0.05$ .

The final adjusted model for HTN included the categorized propensity score (as planned) plus cohort, history of hypertension, presence of windows, and use of smokeless tobacco given the association of these four covariates with HTN (Table 3). After adjustment, wood fuel use was not significantly associated with hypertension (adjusted prevalence ratio 0.76 [95% CI 0.49, 1.17]). A history of hypertension continued to confer and increased risk for HTN at delivery (adjusted prevalence ratio of 4.09 [95%CI 2.80, 5.98]). The presence of windows was associated with a reduction in HTN risk (adjusted prevalence ratio of 0.64 [95% CI 0.49, 0.83]) as was use of smokeless tobacco (adjusted prevalence ratio of 0.71 [0.51, 0.99]).

Linear regression models evaluating the relationship between fuel use and blood pressure measures were subsequently adjusted for the covariates identified in the modeling exercise for HTN at delivery. The association of wood fuel use with a small reduction in DBP and MAP ( $< 5$  mmHg) but not SBP at the time of delivery persisted after adjustment (Table 4).

## **DISCUSSION**

Wood users were one-third less likely to have postpartum blood pressures in the hypertensive range compared with women cooking primarily with gas although this difference did not reach statistical significance. Adjusting for a propensity score that accounted for numerous socioeconomic differences between wood and gas users did little to alter the effect size but served to widen the confidence interval. We did observe that wood fuel users had on average lower mean arterial pressure and diastolic blood pressure at delivery compared to women cooking with gas although this effect size was small ( $< 5$  mmHg) and likely not clinically relevant. Of note, the frequency of a chronic hypertension history was similar between the two groups so this difference does not appear to be driven by a difference in underlying chronic hypertension. The direction of our observed results, even if missing statistical significance, underscores that the link between biomass smoke

exposure and an increased risk for hypertension reported from nonpregnant populations<sup>14–19</sup> may not translate into a similar effect in a pregnant population. Gestational hypertension and preeclampsia are differentially mediated when compared with chronic hypertension. In chronic hypertension, activation of the sympathetic nervous system and the renin-angiotensin-aldosterone system are implicated in pathogenesis<sup>41</sup>; whereas the placenta, clearly absent in nonpregnant individuals, plays a critical role in the development of hypertensive complications of pregnancy. Higher circulating levels of anti-angiogenic factors released from the placenta have been found in women who eventually manifest preeclampsia in their pregnancy; the elevation of these factors may result in widespread endothelial dysfunction with maternal hypertension as one possible manifestation.<sup>42,43</sup>

It remains plausible that biomass smoke might protect against the development of gestational hypertension or preeclampsia. The combustion byproducts of tobacco and biomass fuels are quite similar and cigarette smoking during pregnancy has been consistently associated with a significantly reduced risk for preeclampsia with a pooled OR of 0.51 [95% CI 0.37, 0.63] reported in meta-analysis.<sup>44</sup> Using a Swedish birth registry of over 600,000 births, epidemiologists reported that smokers but not snuff users had a reduced risk for preeclampsia, concluding that cigarette combustion byproducts rather than nicotine itself were responsible for the protection.<sup>46</sup> In laboratory experiments, placental cells incubated in the presence of cigarette smoke release less soluble fms-like tyrosine kinase and preserve the release of placental growth factor<sup>45</sup>, results consistent with a pro-angiogenic state and opposite to the increase in anti-angiogenic factors observed in women that develop preeclampsia.<sup>47</sup> Levels of circulating angiogenic markers have not been evaluated in a population of pregnant women exposed to biomass cook smoke.

There are limitations of our study design. Our measurements were limited to those obtained postpartum and we recognize that blood pressure can return to antepartum values following delivery; if anything, this likely biases our results towards the null. Furthermore, as this was a secondary analysis of a cohort recruited to evaluate the prevalence of malaria during pregnancy, measures obtained were not optimized for the diagnosis of gestational hypertension or preeclampsia. We did not have access to urine specimens for the measurement of proteinuria or laboratory results to evaluate end organ involvement in the majority of subjects. Blood pressure measurements from antenatal visits were not available for review and measurements during labor not consistently taken or recorded. Taken together, whether the hypertension we observed represents preeclampsia, gestational hypertension, underlying chronic hypertension or some combination of these is not clear. Moreover, despite our best efforts to account for differences between wood and gas users with the propensity score model, residual confounding cannot be excluded. For example, women cooking with wood fuel may live in more rural locations and be subject to less ambient air pollution from traffic sources than those cooking with gas.

Primary household fuel may imperfectly represent exposure to household air pollution. We had information on only a few variables related to cooking activity. We lacked information on the location of the kitchen (inside versus outside), other fuels used, or other sources of smoke such as trash burning, incense, mosquito coils, and kerosene lamps. Ambient air pollution levels were not available for this cohort. These unexamined factors contribute to a



woman's cumulative exposure to household air pollution and are unlikely reflected in our dichotomous exposure variable (self-reported wood fuel use versus gas use). Interestingly, the presence of windows, which may represent improved indoor ventilation, was significantly associated with a reduction in HTN prevalence in the adjusted model. The finding underscores that further work is required to understand whether an association of cooking smoke and HTN in pregnancy exists and in what direction the effect lies. Study designs that include repeated measurements of personal exposure to air pollutants during such as carbon monoxide and fine particulate matter would improve our ability to classify exposure.

Our analysis is hypothesis-generating, highlighting the need to clarify whether an association between hypertensive disorders of pregnancy and cooking smoke exists. There is growing international momentum among advocacy organizations and governments to promote reductions in household air pollution from cooking with biomass fuels ([www.cleancookstoves.org/the-alliance](http://www.cleancookstoves.org/the-alliance)). Several randomized improved stove trials are underway that specifically target pregnant women (Nepal [Clinicaltrials.gov #NCT00786877](https://clinicaltrials.gov/ct2/show/study/NCT00786877), Ghana [clinicaltrials.gov #NCT01335490](https://clinicaltrials.gov/ct2/show/study/NCT01335490)). Researchers should enumerate what return on investment is to be expected for not only infant but also maternal outcomes. Particular attention should be paid to evaluating whether reductions in cook smoke translate into an altered risk for HTN in pregnancy as policymakers will want to know whether reductions in household air pollution during pregnancy will benefit the mother as well as the infant.

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## REFERENCES

1. Pachauri, S.; Brew-Hammond, A.; Barnes, DF.; Bouille, DH.; Gitonga, S.; Modi, V., et al. Energy access for development. In: Johansson, TB.; Nakicenovic, N.; Patwardhan, A.; Gomez-Echeverri, L., editors. *Global Energy Assessment: Toward a Sustainable Future*. New York: Cambridge University Press; 2012. p. 1401-1458.
2. Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani K, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet*. 2012; 380(9859):2224–2260. [PubMed: 23245609]
3. World Health Organization. [Accessed April 18, 2014] Household air pollution and health: WHO fact sheet. Updated March 2014. Available online at <http://www.who.int/mediacentre/factsheets/fs292/en/>.
4. Nairobi, Kenya: United Nations: United Nations Development Program-World Health Organization Joint Report 2009: The energy access situation in developing countries: A review focused on least developed countries in sub-Saharan Africa. [<http://www.who.int/indoorair/publications/energyaccesssituation/en/>]

5. Mavalankar DV, Gray RH, Trivedi CR. Risk factors for preterm and term low birthweight in Ahmedabad, India. *Int J Epidemiol.* 1992; 21(2):263–272. [PubMed: 1428479]
6. Boy E, Bruce N, Delgado H. Birthweight and exposure to kitchen wood smoke during pregnancy in rural Guatemala. *Environ Health Perspect.* 2002; 110(1):109–114. [PubMed: 11781172]
7. Mishra V, Dai X, Smith K, Mika L. Maternal exposure to biomass smoke and reduced birth weight in Zimbabwe. *Ann Epidemiol.* 2004; 14(10):740–747. [PubMed: 15519895]
8. Mishra V, Retherford RD, Smith KR. Cooking smoke and tobacco smoke as risk factors for stillbirth. *Int J Env Health Res.* 2005; 15(6):397–410. [PubMed: 16506434]
9. Siddiquai AR, Gold EB, Yang X, Lee K, Brown KH, Bhutta ZA. Prenatal exposure to wood fuel smoke and low birth weight. *Environ Health Perspect.* 2008; 116:543–549.
10. Tielsch JM, Katz J, Thulasirai RD, Thulasiraj RD, Coles CL, Sheeladevi S, et al. Exposure to indoor biomass fuel and tobacco smoke and risk of adverse reproductive outcomes, mortality, respiratory morbidity and growth among newborn infants in south India. *Int J Epidemiol.* 2009; 38(5):1351–1363. [PubMed: 19759098]
11. Epstein MB, Bates MN, Arora NK, Balakrishnan K, Jack DW, Smith KR. Household fuels, low birth weight, and neonatal death in India: The separate impacts of biomass, kerosene, and coal. *Int J Hyg Environ Health.* 2013; 216(5):523–532. [PubMed: 23347967]
12. Pope DP, Mishra V, Thompson L, Siddiqui AR, Rehfuess EA, Weber M, et al. Risk of low birth weight and stillbirth associated with indoor air pollution from solid fuel use in developing countries. *Epidemiol Rev.* 2010; 32(1):70–81. [PubMed: 20378629]
13. Wylie BJ, Coull BA, Hamer DH, Singh MP, Jack D, Yeboah-Antwi K, et al. Impact of biomass fuels on pregnancy outcomes in central East India. *Environ Health.* 2014; 13(1):1. [PubMed: 24405644]
14. Baumgartner J, Schauer JJ, Ezzati M, Lu L, Cheng C, Patz JA, et al. Indoor air pollution and blood pressure in adult women living in China. *Environ Health Perspect.* 2011; 119(10):1390–1395. [PubMed: 21724522]
15. Clark ML, Bazemore H, Reynolds SJ, Heiderscheidt JM, Conway S, Bachand AM, et al. A baseline evaluation of traditional cook stove smoke exposures and indicators of cardiovascular and respiratory health among Nicaraguan women. *Int J Occ Environ Health.* 2011; 17(2):113–121.
16. Dutta A, Mukherjee B, Das D, Banerjee A, Ray MR. Hypertension with elevated levels of oxidized and low-density lipoprotein and anticardiolipin antibody in the circulation of premenopausal Indian women chronically exposed to biomass smoke during cooking. *Indoor Air.* 2011; 21(2): 165–176. [PubMed: 21118307]
17. Dutta A, Ray MR. Prevalence of hypertension and pre-hypertension in rural women: a report from the villages of West Bengal, a state in the eastern part of India. *Austr J Rural Health.* 2012; 20(4): 219–225.
18. Lee MS, Hang JQ, Zhang FY, Dai HL, Su L, Christiani DC. In-home solid fuel use and cardiovascular disease: a cross-sectional analysis of the Shanghai Putuo study. *Env Health.* 2012; 11:18. [PubMed: 22455369]
19. Arbex MA, Saldiva PH, Pereira LA, Braga AL. Impact of outdoor biomass air pollution on hypertension hospital admissions. *J Epidemiol Community Health.* 2010; 64(7):573–579. [PubMed: 20466708]
20. McCracken JP, Smith KR, Diaz A, Mittleman MA, Schwartz J. Chimney stove intervention to reduce long-term wood smoke exposure lowers blood pressure among Guatemalan women. *Environ Health Perspect.* 2007; 115(7):996–1001. [PubMed: 17637912]
21. Clark ML, Bachand AM, Heiderscheidt JM, Yoder SA, Luna B, Volckens J, et al. Impact of a cleaner-burning cookstove intervention on blood pressure in Nicaraguan women. *Indoor Air.* 2013; 23(2):105–114. [PubMed: 22913364]
22. Vigh M, Yuneisan M, Shariat M, Niroomanesh S, Ramezanzadeh F. Environmental carbon monoxide related to pregnancy hypertension. *Women Health.* 2011; 51(8):724–738. [PubMed: 22185288]
23. Mobasher Z, Slaam MT, Goodwin TM, Lurmann F, Ingles SA, Wilson ML. Associations between ambient air pollution and hypertensive disorders of pregnancy. *Environ Res.* 2013; 123:9–16. [PubMed: 23522615]

24. Xu X, Hu H, Ha S, Roth J. Ambient air pollution and hypertensive disorder of pregnancy. *J Epidemiol Community Health*. 2014; 68(1):13–20. [PubMed: 24022815]
25. Dadvand P, Figueras F, Basagana X, Beelen R, Martinez D, Cirach M, et al. Ambient air pollution and preeclampsia: a spatiotemporal analysis. *Environ Health Perspect*. 2013; 121(11–12):1365–1371. [PubMed: 24021707]
26. Lee PC, Roberts JM, Catov JM, Talbott EO, Ritz B. First trimester exposure to ambient air pollution, pregnancy outcomes, and adverse birth outcomes in Allegheny County, PA. *Matern Child Health J*. 2013; 17(3):545–555. [PubMed: 22544506]
27. Wu J, Wilhelm M, Chung J, Ritz B. Comparing exposure assessment methods for traffic-related air pollution in an adverse pregnancy outcome study. *Environ Res*. 2011; 111(5):685–692. [PubMed: 21453913]
28. Jedrychowski WA, Pererea FP, Maugeri U, Spengler J, Mroz E, Flak E, et al. Prohypertensive effect of gestational personal exposure to fine particulate matter: prospective cohort study in non-smoking and non-obese pregnant women. *Cardiovasc Toxicol*. 2012; 12(3):216–225. [PubMed: 22328329]
29. Zhai D, Guo Y, Smith G, Krewski D, Walker M, Wen SW. Maternal exposure to moderate ambient carbon monoxide is associated with decreased risk of preeclampsia. *Am J Obstet Gynecol*. 2012; 207(1):57. [PubMed: 22521459]
30. Van de Hoeven EH, Jaddoe VW, de Kluizenaar Y, Hofman A, Mackenbach JP, Steegers EA, et al. Residential traffic exposure and pregnancy-related outcomes: a prospective birth cohort study. *Environ Health*. 2009; 8:59. [PubMed: 20028508]
31. Conde-Agudelo A, Althabe F, Belizan JM, Kafury-Goeta AC. Cigarette smoking during pregnancy and risk of preeclampsia: a systematic review. *Am J Obstet Gynecol*. 1999; 181(4):1026–1035. [PubMed: 10521771]
32. Kassenbaum NJ, Bertozzi-Villa A, Coggeshall MS, Shackelford KA, Steiner C, Heuton KR, et al. Global, regional and national levels and causes of maternal mortality during 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet*. 2014; 384(9947):980–1004. [PubMed: 24797575]
33. Rosenbaum P, Rubin DB. The central role of the propensity score in observational studies for causal effects. *Biometrika*. 1983; 70(1):41–55.
34. Hamer DH, Singh MP, Wylie BJ, Yeboah-Antwi K, Tuchman J, Desai M, et al. Burden of malaria in pregnancy in Jharkhand State, India. *Malar J*. 2009; 8:210. [PubMed: 19728882]
35. Singh N, Singh MP, Wylie BJ, Hussain M, Kojo YA, Shekhar C, et al. Malaria prevalence among pregnant women in two districts with differing endemicity in Chhattisgarh, India. *Malar J*. 2012; 11:274. [PubMed: 22882903]
36. Sabin LL, Rizal A, Brooks MI, Singh MP, Tuchman J, Wylie BJ, Joyce KM, Yeboah-Antwi K, Singh N, Hamer DH. Attitudes, knowledge, and practices regarding malaria prevention and treatment among pregnant women in Eastern India. *Am J Trop Med Hyg*. 2010; 82(6):1010–1016. [PubMed: 20519593]
37. Wylie BJ, Hashmi AH, Singh N, Tuchman J, Hussain M, Sabin L, et al. Availability and utilization of malaria prevention strategies in pregnancy in eastern India. *BMC Public Health*. 2010; 10:557. [PubMed: 20849590]
38. Bhattacharya SC, Albina DO, Abdul Salam P. Emission factors of wood and charcoal-fired cookstoves. *Biomass Bioenergy*. 2002; 23(6):453–469.
39. Lam NL, Smith KR, Gauthier A, Bates MN. Kerosene: a review of household uses and their hazards in low-and middle-income countries. *J Toxicol Env Health B Crit Rev*. 2012; 15(6):396–432. [PubMed: 22934567]
40. Ezzati M, Mbinda BM, Kammen DM. Comparison of emissions and residential exposure from traditional and improved cookstoves in Kenya. *Env Science Techn*. 2000; 34(4):578–583.
41. Oparil S, Zaman MA, Calhoun DA. Pathogenesis of hypertension. *Ann Intern Med*. 2003; 139:761–776. [PubMed: 14597461]
42. Lain KY, Roberts JM. Contemporary concepts of the pathogenesis and management of preeclampsia. *JAMA*. 2002; 287(24):3183–3186. [PubMed: 12076198]

43. Frishman WH, Schlocker SJ, Awad K, Tejani N. Pathophysiology and medical management of systemic hypertension in pregnancy. *Cardiol Rev.* 2005; 13(6):274–284. [PubMed: 16230884]
44. Castles A, Adams EK, Melvin CL, Kelsch C, Boulton ML. Effects of smoking during pregnancy: Five meta-analyses. *Am J Prev Med.* 1999; 16(3):208–215. [PubMed: 10198660]
45. Mehendale R, Hibbard J, Fazleabas A, Leach R. Placental angiogenesis markers sFLT-1 and PlGF: Response to cigarette smoke. *Am J Obstet Gynecol.* 2007; 197(4):363. e1–363. e5. [PubMed: 17904960]
46. Wikstrom AK, Stephansson O, Cnattingius S. Tobacco use during pregnancy and preeclampsia risk: effect of cigarette smoking and snuff. *Hypertension.* 2010; 55(5):1254–1259. [PubMed: 20231527]
47. Levine RJ, Maynard SE, Qian C, Lim KH, England LJ, Yu KF, et al. Circulating angiogenic factors and the risk of preeclampsia. *N Eng J Med.* 2004; 350(7):672–683.

**Table 1**

Demographic, obstetric and socioeconomic variables in pregnant women cooking with wood versus gas

	Wood group <sup>a</sup> n=1134	Gas group <sup>a</sup> n=235	Significance p-value
<i>Cohort<sup>b</sup></i>			
Jharkhand	389 (34.3%)	80 (34.0%)	0.9389
Chhattisgarh	745 (65.7%)	155 (66.0%)	
<i>Time spent cooking</i>			
Upper decile of daily cook time	202 (17.8%)	21 (8.9%)	0.0008
<i>Ventilation</i>			
House has windows <sup>b</sup>	749 (66.1%)	211 (89.8%)	<0.0001
<i>Maternal and pregnancy characteristics</i>			
Age < 20 years <sup>c</sup>	88 (7.8%)	10 (4.3%)	0.0579
Overweight (BMI ≥ 25)	17 (1.5%)	8 (3.4%)	0.0471
Underweight (BMI < 18.5)	196 (17.3%)	29 (12.3%)	0.0627
Primiparous	602 (53.1%)	123 (52.3%)	0.8348
Multiple gestation	14 (1.2%)	1 (0.4%)	0.4900
History of hypertension <sup>b</sup>	14 (1.2%)	3 (1.3%)	1.000
<i>Maternal habits</i>			
Smokes	2 (0.2%)	0 (0%)	1.000
Use of smokeless tobacco <sup>b</sup>	328 (28.9%)	11 (4.7%)	<0.0001
Drinks alcohol	23 (2.0%)	1 (0.4%)	0.1034
<i>Medical and obstetric history</i>			
Adequate antenatal visits (≥ 4)	381 (33.8%)	146 (62.1%)	<0.0001
Taking iron	870 (76.7%)	206 (87.7%)	0.0002
Taking folate	817 (72.1%)	197 (83.8%)	0.0002
History of diabetes	2 (0.2%)	0 (0%)	1.000
History of hypertension <sup>b</sup>	14 (1.2%)	3 (1.3%)	0.9578
Multiple gestation	14 (1.2%)	1 (0.4%)	0.2782
Placental or peripheral parasitemia at delivery	42 (3.7%)	7 (3.0%)	0.5724
<i>Socio-demographic characteristics<sup>d</sup></i>			
Married	1132 (99.8%)	235 (100.0%)	1.0000
Historically disadvantaged caste <sup>e</sup>	992 (87.6%)	133 (56.6%)	<0.0001
Agricultural work	266 (23.5%)	7 (3.0%)	<0.0001
Formal schooling ≥ 5 years	643 (56.7%)	40 (17.02%)	<0.0001
Impermanent/semi-permanent roof	1095 (96.6%)	93 (39.67%)	<0.0001
Impermanent/semi-permanent floor	991 (87.4%)	34 (14.5%)	<0.0001
Impermanent/semi-permanent wall	1000 (88.2%)	37 (15.7%)	<0.0001
Owns radio	267 (23.5%)	128 (54.5%)	<0.0001

	Wood group <sup>a</sup> n=1134	Gas group <sup>a</sup> n=235	Significance p-value
Owens electric fan	364 (32.1%)	214 (91.1%)	<0.0001
Owens room cooler	45 (4.0%)	120 (51.1%)	<0.0001
Owens television <sup>b</sup>	363 (32.0%)	210 (89.4%)	<0.0001
Owens refrigerator	6 (0.5%)	70 (29.8%)	<0.0001
Owens motorcycle	131 (11.6%)	147 (62.6%)	<0.0001
Owens 4 wheel vehicle	14 (1.2%)	29 (12.3%)	<0.0001
Propensity score <sup>f</sup>	0.93 (±0.14)	0.32 (±0.32)	<0.0001

<sup>a</sup>Values represent n (%) or mean (± standard deviation).

<sup>b</sup>Significantly associated with hypertension (p <0.05)

<sup>c</sup>Age categorized as many women unable to recall their birth date.

<sup>d</sup>Considered for inclusion in propensity score model.

<sup>e</sup>Historically disadvantaged castes include Scheduled Caste, Other Backward Caste, and Scheduled Tribes.

<sup>f</sup>Propensity score model: Propensity to use wood=0.3155 + (0.76896\*impermanent walls) + (0.3473\*impermanent floors) + (0.6156\*impermanent roof) + (0.3952\*member of historically disadvantaged caste) + (0.2099\*primary school education or less) + (0.5277\*agricultural occupation) – (0.0444\* owns radio) – (0.5265\*owns electric fan) – (0.6554\*owns room cooler) – (0.1104\*owns television) – (0.5265\*owns refrigerator) + (0.0923\*owns 4 wheel vehicle).

**Table 2**

Association of wood fuel use with maternal hypertension and blood pressure at delivery, unadjusted analyses

	Wood group <sup>a</sup> n=1134	Gas group <sup>a</sup> n=235	Unadjusted effect size [95% CI]
HTN	166 (14.6%)	46 (19.6%)	0.75 [0.56, 1.00] <sup>b</sup>
Severe HTN	10 (0.9%)	2 (0.9%)	1.04 [0.22, 9.79] <sup>c</sup>
SBP (mmHg)	116.3 (±10.5)	117.0 (±9.2)	- 0.7 [-2.1, 0.7] <sup>d</sup>
DBP (mmHg)	76.6 (±8.7)	78.6 (±8.3)	-2.0 [-3.3, -0.8] <sup>d</sup>
MAP (mmHg)	89.8 (±8.5)	91.4 (±7.5)	-1.6 [-2.7, -0.5] <sup>d</sup>

CI= confidence interval. DBP= diastolic blood pressure. HTN= hypertension. MAP= mean arterial pressure. SBP=systolic blood pressure.

<sup>a</sup>Values represent n (%) or mean (± standard deviation).

<sup>b</sup>Effect size represents prevalence ratio estimated with log-binomial regression.

<sup>c</sup>Effect size represent odds ratio, estimated with exact logistic regression, given rarity of event.

<sup>d</sup>Effect size represents beta (in mmHg) estimated with linear regression.

**Table 3**

Adjusted association of maternal hypertension at delivery with wood fuel use and other risk factors

	Women with HTN <sup>a</sup> n=212	Normotensive women <sup>a</sup> n=1157	Unadjusted effect size <sup>b</sup> [95% CI]	Adjusted effect size <sup>c</sup> [95% CI]
Wood fuel use	166 (78.3%)	968 (83.66%)	0.75 [0.56, 1.00]	0.76 [0.49, 1.17]
Cohort <sup>d</sup>	87 (41.0%)	382 (33.0%)	1.33 [1.04, 1.71]	1.03 [0.80, 1.33]
Jharkhand	125 (59.0%)	775 (67.0%)		
Chhattisgarh				
History of hypertension	12 (5.7%)	5 (0.4%)	4.77 [3.42, 6.65]	4.09 [2.80, 5.98]
House has windows	123 (58.0%)	837 (72.3%)	0.59 [0.46, 0.75]	0.64 [0.49, 0.83]
Use of smokeless tobacco	35 (16.5%)	304 (26.3%)	0.60 [0.43, 0.84]	0.71 [0.51, 0.99]
Upper decile of time spent cooking	30 (14.2%)	193 (16.7%)	0.84 [0.59, 1.21]	----

HTN=hypertension (systolic blood pressure &gt; 139 mmHg or diastolic blood pressure &gt; 89 mmHg). CI= confidence interval.

<sup>a</sup> Values represent n(%).<sup>b</sup> Effect size represents prevalence ratio, estimated by log-binomial regression.<sup>c</sup> Covariates included in adjusted model include wood fuel use, categorized propensity score, cohort, history of hypertension, house with windows and use of smokeless tobacco.<sup>d</sup> Chhattisgarh as referent.



**Table 4**

Adjusted association of wood fuel use with measured maternal blood pressure at delivery

	Adjusted effect size <sup>a</sup> [95% CI]
SBP (mmHg)	-1.79 [-3.93, 0.26]
DBP (mmHg)	-2.04 [-3.77, -0.31]
MAP (mmHg)	-1.96 [-3.60, -0.30]

CI= confidence interval. DBP= diastolic blood pressure. MAP=mean arterial pressure. SBP= systolic blood pressure.

<sup>a</sup>Effect size represents beta in mmHg estimated by linear regression adjusted for cohort, windows, smokeless tobacco, and history of hypertension.

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