Published in final edited form as:
Respir Med. 2006 July ; 100(7): 1208–1215. doi:10.1016/j.rmed.2005.10.020.

Relationship of pulmonary function among women and children to indoor air pollution from biomass use in rural Ecuador

Seppo T. Rinne[a], **Edgar J. Rodas**[b], **Brooke S. Bender**[c], **Mikael L. Rinne**[d], **Joshua M. Simpson**[e], **Regina Galer-Unti**[f], and **Larry T. Glickman**[a,*]

[a]Department of Veterinary Pathobiology, School of Veterinary Medicine, Purdue University, West Lafayette, IN 47907, USA

[b]Cinterandes Foundation, Faculty of Medicine, University of Azuay, Cuenca, Ecuador

[c]Emergency Department, Jacobi Hospital, Albert Einstein School of Medicine, New York, NY, USA

[d]School of Medicine, Indiana University, Indianapolis, IN, USA

[e]Center for Environmental Research and Conservation, Columbia University, New York, NY, USA

[f]Department of Health and Kinesiology, Purdue University, West Lafayette, IN 47907, USA

Summary

Approximately half the world uses biomass fuel for domestic energy, resulting in widespread exposure to indoor air pollution (IAP) from biomass smoke. IAP has been associated with many respiratory diseases, though it is not clear what relationship exists between biomass use and pulmonary function. Four groups containing 20 households each were selected in Santa Ana, Ecuador based on the relative amount of liquid petroleum gas and biomass fuel that they used for cooking. Pulmonary function tests were conducted on each available member of the households ≥7 years of age. The pulmonary functions of both children (7–15 years) and women (≥16 years) were then compared between cooking fuel categories using multivariate linear regression, controlling for the effects of age, gender, height, and exposure to tobacco smoke. Among the 80 households, 77 children and 91 women performed acceptable and reproducible spirometry. In multivariate analysis, children living in homes that use biomass fuel and children exposed to environmental tobacco smoke had lower forced vital capacity and lower forced expiratory volume in 1 s (P<0:05). However, no significant difference in pulmonary function was observed among women in different cooking categories. Results of this study demonstrate the harmful effects of IAP from biomass smoke on the lung function of children and emphasize the need for public health efforts to decrease exposure to biomass smoke.

Keywords

Pulmonary function tests; Biomass fuel; Indoor air pollution; Ecuador

Introduction

Indoor air pollution (IAP) is the second largest environmental contributor to morbidity worldwide, surpassed only by unsafe water and sanitation.[1] The largest source of IAP is the combustion of biomass fuels (wood, charcoal, crop residues, or dung) for domestic energy

*Corresponding author. Tel.: +1 765 494 2294; fax: +1 765 494 9830. ltg@purdue.edu (L.T. Glickman).

by nearly half the world's population. [2] IAP from biomass combustion has been associated with many common diseases including acute lower respiratory infections (ALRI), middle ear infections (otitis media), cancers of the nasopharynx and larynx, perinatal disease, and diseases of the eye (cataracts and blindness).[3] It is estimated that IAP causes 2.2–2.8 million deaths annually including approximately 1 million deaths due to ALRI among children.[4,5] Women and children are particularly vulnerable to these adverse health effects because of their greater exposure to IAP from cooking.

Exposure to environmental tobacco smoke (ETS) is known to cause decreased pulmonary function and abnormal lung development, particularly with prenatal and early childhood exposures.[6] Several studies also suggest that exposure to biomass smoke is associated with impaired pulmonary function.[7–13] Many of these studies, however, did not control for the effects of age and anthropometric factors such as height.[7,9,10]

The primary goal of this study was to examine the impact of biomass fuel use on pulmonary function among women and children in a rural Ecuadorian community. We hypothesized a significant decrease in pulmonary function with increasing use of biomass fuel among both women and children. The study was conducted in Santa Ana, Ecuador due to the extensive use of biomass fuel and a high prevalence of respiratory disease. This study was a collaborative effort between Purdue University and The Cinterandes Foundation.

Materials and methods

Study site

Santa Ana is a rural community in the Andes of southern Ecuador. It is located 18 km southeast of the city of Cuenca and extends across 46 km of mountainous countryside at altitudes range from 2300 to 3200 m. Climate in the region is generally cool and dry with average monthly lows of 8 °C and highs of 20 °C. The study took place during the dry season from June to November of 2004.

There are approximately 5000 people in Santa Ana living in nearly 1000 households. Eighty-five percents of the population are mestizos (of mixed European and Native American ancestry) while the remaining 15% are Quechuan (a Native American population). Most community members work in manual labor and almost all households rely on sustenance farming. The illiteracy rate is high (14%) and only 30% of adults have completed elementary education. Though nearly 80% of households receive electricity, the majority lack municipal water and many do not have access to latrine or toilet facilities. Most households rely on biomass fuel for cooking, which is typically done indoors over open wood fires. Some households also rely on liquid petroleum gas (LPG) stoves for part or all of their cooking. Exposure to LPG has been associated with lower pulmonary function when compared with a cleaner fuel such as electricity.[14–16] However, LPG is known to be a much cleaner fuel source than biomass combustion, and at this time none of the households in Santa Ana used electricity for cooking. All houses studied had a separate room for cooking, but ventilation was poor with few windows and doors, which were usually kept closed.

Sampling and data collection

Households were selected by random and quota sampling. All households in the community were initially randomized by assigning each a random number using a random number generator. Households were then sequentially visited based on their random number. The cooking fuel source was identified for each of the households visited, and the interviewer used quota sampling to identify four cooking categories that contained 20 households each. The four categories were: LPG only, primarily LPG with some biomass fuel, primarily biomass fuel with some LPG, and biomass fuel only. If a household declined to participate, the next household on the randomly generated list was contacted.

The study was initially explained to local government, church, and healthcare workers in Santa Ana and their oral consent was obtained. Written consent for each child's participation was obtained in Spanish from an adult in the household and written assent was obtained from literate children. Households were visited by a trained healthcare worker and an outreach member of the local government. The age, gender, height, and exposure to ETS were recorded for each woman and child living in the household. Pulmonary function tests were conducted on individuals ≥7 years old using a Koko Trek portable flow spirometer (Ferraris Respiratory, Louisville, Colorado) that was calibrated daily with a 3 L syringe. Spirometric measurements included forced vital capacity (FVC), forced expiratory volume in 1 s (FEV_1), FEV_1/FVC (expressed as a percentage), and forced expiratory flow over the middle half of the FVC ($FEF_{25–75\%}$). Spirometry was conducted with subjects in a standing position with a nose clip applied. Based on guidelines of the American Thoracic Society, maneuvers were only accepted if they had low back-extrapolated volume (<5% of the FVC and <0.15 L), both the FVC and FEV_1 for were within 0.20 L of the best effort FVC and FEV_1, and there was a low volume accumulated at the end of the effort.[17] Subject performed two acceptable and reproducible efforts. Data analysis was conducted on the largest FVC of the two curves, the largest FEV_1, the ratio of the largest FEV_1 to the largest FVC, and the $FEF_{25–75\%}$ from the curve with the largest FVC+FEV_1 sum.

Statistics

Analysis of variance (ANOVA) and Pearson's χ^2 tests were used to compare the difference in baseline characteristics (age, gender, and height) between cooking fuel categories for both women (≥16 years old) and children (7–15 years old). Multivariate linear regression analysis was used to assess the association of cooking fuel with pulmonary function, controlling for age, gender, height, and exposure to tobacco smoke (defined by the presence of a smoker in the household). A *P*-value <0.05 was considered significant for all analyses. Statistical analyses were performed using SPSS version 12.0.[18]

All of the research procedures were approved by the Committee on the Use of Human Research Subjects at Purdue University.

Results

Among the 80 households in the study, 77 children (of 88 available children) and 91 women (of 114 available women) performed acceptable and reproducible spirometry. There were no significant differences in gender, age, height, or exposure to ETS between children in different cooking fuel categories (Table 1). Among women, there were significant differences in the mean height ($P = 0:02$) of adults in different cooking fuel categories. Women cooking with LPG were taller than those cooking with biomass fuels. Women living in households that cooked with LPG also tended to be younger than women living in households that cooked with biomass fuel ($P = 0:07$). Among households in the study, only adult men smoked tobacco. The median number of cigarettes smoked by these men was 3.5/day (Interquartile Range = 8).

Mean pulmonary function values are presented for women and children in Table 2. Multivariate analysis for pulmonary function of children showed significantly lower FVC among children living in households that cooked with biomass fuel only ($P = 0:04$), primarily with biomass ($P = 0:03$), and primarily LPG ($P = 0:01$) when compared with children living in households that cooked with LPG only (Table 3). Similarly, a lower FEV_1 was observed among children living in households that cooked with biomass fuel only ($P =$

0:05), primarily with biomass fuel ($P = 0{:}01$) and primarily with LPG ($P = 0{:}02$). Passive exposure to tobacco smoke (defined by the presence of a smoker in the household) was also associated with a significantly lower FVC ($P = 0{:}01$) and FEV_1 ($P = 0{:}001$) among children. No significant differences in FEV_1/FVC or $FEF_{25–75\%}$ were noted between children in different cooking categories or children exposed to passive tobacco smoke. Furthermore, there were no significant differences in pulmonary function among women in different cooking fuel categories (Table 4).

Discussion

This study documents lower pulmonary function among children living in households that cook with biomass fuel and children exposed to ETS. No difference in pulmonary function, however, was observed among women in different cooking fuel categories. The mechanism for decreased pulmonary function and impaired lung development among children exposed to air pollutants is not clear. Fetal and neonatal exposures to pollutants may alter lung "programming" causing permanent changes in lung structure and function.[19] The pathogenesis of these changes could be related to inflammation, thickened airway walls, small caliber airways, increased smooth muscle tone, or decreased elastic recoil.[20]

Exposure to ETS

Among children, exposure to ETS is known to decrease pulmonary function and impair lung growth.[6] Fetal and early postnatal exposure to maternal smoking has been associated with significant deficits in lung function that persist into young adulthood.[21–23] Childhood exposure to ETS has also been implicated in lung damage and reduced lung function. Studies on ETS exposure among children often show a lower FEV_1 when compared to non-exposed children.[24–27] The impact of ETS on lung volume, however, is less clear. Though many studies show decreased FVC among children exposed to ETS,[24,28,29] the majority find no significant difference between exposed and non-exposed children.[6] Children exposed to ETS in our study were found to have both lower FEV_1 and FVC.

Fewer studies have examined the association between pulmonary function and ETS in adults, and these often produce inconsistent results.[30] Some studies demonstrated lower lung flow (FEV_1 and $FEF_{25–75\%}$) among adults exposed to ETS,[31,32] while others found no significant difference.[33–35] Though adults exposed to ETS may have slight changes in pulmonary function, ETS alone is not expected to have clinically significant impacts on adult lung function.[36] The fact that we did not see a significant functional deficit with exposure to ETS among women may also be due to a recent onset of smoking in the community and the small number of cigarettes smoked by the smokers in our study.

Exposure to biomass smoke

Previous studies have also demonstrated lower pulmonary function among children exposed to biomass smoke.[7,9,10]

Jordanian school children living in homes that cooked with wood or kerosene had significantly lower FVC (1.02 vs. 1.32 L), FEV_1 (0.91 vs. 1.24 L), and $FEF_{25–75\%}$ (1.24 vs. 1.86 L/s) than children living in homes that cooked with electricity.[9] The same study also showed that children exposed to ETS had lower FVC (1.29 vs. 1.49 L), FEV_1 (1.20 vs. 1.40 L), and $FEF_{25–75\%}$ (1.84 vs. 2.24 L) when compared with non-exposed children. In northern India, boys living in houses that cooked with biomass fuel had significantly lower mean FVC and FEV_1 (2.40 and 2.19 L, respectively) when compared with boys living in homes that cooked with kerosene (2.41 and 2.23 L), mixed fuels (2.48 and 2.33 L), or LPG (3.03 and 2.75 L).[7] School children in southern Turkey also had decreased FVC and FEV_1 with exposure to wood-burning stoves.[10] These studies consistently demonstrate decreased pulmonary function among children exposed to IAP from biomass smoke. However, the analysis of these studies did not control for the confounding effects of age or height. The present study found similar relationships, and also controlled for the effects of age, gender, height, and ETS, in multivariate analysis.

Adults exposed to IAP from biomass combustion have also been found to have decreased pulmonary function, often associated with COPD.[3] COPD is usually attributed to tobacco smoke, which accounts for more than 80% of cases in the developed world.[37] In developing countries, however, COPD is prevalent in areas where smoking is uncommon. Numerous studies have identified biomass smoke as a primary risk factor for COPD in rural areas.[8,38–41] For example, in Bogotá, Colombia hospital patients that cooked with firewood were 3.9 times as likely to have severe obstructive pulmonary disease than patients who did not cook with wood.[39] Other hospital-based investigations found adjusted odds ratios for COPD in the range of 1.8–9.7 associated with exposure to biomass smoke.[3]

Observational studies of non-smoking women with long-term exposures to biomass smoke have shown combined restrictive and obstructive changes in pulmonary function.[11,12,13,42,43] In the Mid-Antolia region of Turkey, adults that used biomass fuel for cooking or heating had significant functional deficiencies (FVC, FEV_1, FEV_1/FVC, $FEF_{25–75\%}$) when compared with adults that did not use biomass fuel.[13] On physical exam, pre-dominant findings of biomass smoke exposure include bilateral basilar crackles and clinical signs of cor pulmonale.[11,12,43] A diffuse reticular-nodular pattern has commonly been observed on radiograph, and histopathologic examination of lung biopsies revealed extensive anthracosis and thickening of the basement membrane. Signs of acute and chronic inflammation were detected with bronchoscopy. Women in theses studies were commonly exposed to biomass smoke for 8 h/day over the previous 50 years.[12]

Women exposed to biomass smoke in our study did not have significantly different lung functions when compared with unexposed women. Santa Ana has recently undergone economic development, and it has only been in the previous 10 years that households began cooking with LPG. This recent change in domestic energy may explain why differences in pulmonary function were not detected among women in different cooking fuel categories. All women were likely to have been raised in households that relied completely on biomass fuel, and thus are expected have similar childhood exposures. Current differences in IAP exposure among women may not have a significant impact, since the adult lung is thought to be less susceptible to the effects of air pollution than children's lungs.[36]

Limitations

Individuals of Quechuan descent have been shown to have lower ventilatory response to sustained hypoxia.[44] Several studies have also shown that Quechuans have increased pulmonary function when compared with individuals of European/North American descent.[45–47] A study comparing the height-adjusted FVC between Quechuan natives and expatriate Europeans/North Americans born and raised at high altitudes showed significantly greater FVC among the native Quechuan.[47] This increased pulmonary function may be associated with an accelerated growth in lung function relative to stature, the large chest sizes of Quechuan people, and an increased alveolar surface area.[48] The findings of this paper may, therefore, be unique to the population that was studied. We did not assess the ancestry of individual participants in our study and, therefore, cannot comment on the impact of ethnicity on pulmonary function among individuals in Santa Ana. Furthermore, there is not an appropriate reference equation to calculate the percent-predicted pulmonary function of individuals in this population.

Major limitations of this study also include its cross-sectional design. Thus, past exposure to biomass smoke and recent changes in biomass use were not assessed. The study was also limited by the use of a surrogate measure (cooking fuel) to assess IAP from biomass smoke, though levels of biomass smoke are expected to increase with increasing use of biomass fuel.[49] Furthermore, we cannot comment on the duration of exposure to different fuel sources as we could not adequately assess cooking time among households. Lastly, the study was limited by its relatively small sample size in each cooking fuel category, decreasing the power to detect small differences in pulmonary function.

Conclusions

This study contributes to the evidence of the adverse impact of IAP from biomass combustion on pulmonary function. Results of the study demonstrated decreased pulmonary function among children living in homes that cook with biomass fuel when compared with children living in homes that cook with LPG only. Prospective studies are needed to assess the long-term effects of biomass smoke on lung development and lung growth among children. Further research is also warranted to assess the potential reversibility of changes in pulmonary function due to childhood exposure to biomass smoke. Given the extensive use of biomass fuels, public health efforts in the developing world that are concerned with respiratory health should address the risks IAP exposure.

References

1. WHO. Reducing risks, promoting healthy life. Genova: World Health Organization; 2002.
2. Smith KR. Indoor air pollution in developing countries: recommendations for research. Indoor Air. 2002; 12(3):198–207. [PubMed: 12244750]
3. Bruce N, Perez-Padilla R, Albalak R. Indoor air pollution in developing countries: a major environmental and public health challenge. Bull WHO. 2000; 78(9):1078–92. [PubMed: 11019457]
4. Schwela D. Exposure to environmental chemicals relevant for respiratory hypersensitivity: global aspects. Toxicol Lett. 1996; 86(2–3):131–42. [PubMed: 8711765]
5. Smith KR, Mehta S. The burden of disease from indoor air pollution in developing countries: comparison of estimates. Int J Hyg Environ Health. 2003; 206(4–5):279–89. [PubMed: 12971683]
6. Cook DG, Strachan DP, Carey IM. Health effects of passive smoking. 9. Parental smoking and spirometric indices in children. Thorax. 1998; 53(10):884–93. [PubMed: 10193379]
7. Behera D, Sood P, Singh S. Passive smoking, domestic fuels and lung function in north Indian children. Indian J Chest Dis Allied Sci. 1998; 40(2):89–98. [PubMed: 9775566]
8. Dossing M, Khan J, al-Rabiah F. Risk factors for chronic obstructive lung disease in Saudi Arabia. Respir Med. 1994; 88(7):519–22. [PubMed: 7972976]
9. Gharaibeh NS. Effects of indoor air pollution on lung function of primary school children in Jordan. Ann Trop Paediatr. 1996; 16(2):97–102. [PubMed: 8790672]
10. Guneser S, Atici A, Alparslan N, Cinaz P. Effects of indoor environmental factors on respiratory systems of children. J Trop Pediatr. 1994; 40(2):114–6. [PubMed: 8015025]
11. Ozbay B, Uzun K, Arslan H, Zehir I. Functional and radiological impairment in women highly exposed to indoor biomass fuels. Respirology. 2001; 6(3):255–8. [PubMed: 11555385]
12. Sandoval J, Salas J, Martinez-Guerra ML, Gomez A, Martinez C, Portales A, et al. Pulmonary arterial hypertension and cor pulmonale associated with chronic domestic woodsmoke inhalation. Chest. 1993; 103(1):12–20. [PubMed: 8417864]
13. Sumer H, Turaclar UT, Onarlioglu T, Ozdemir L, Zwahlen M. The association of biomass fuel combustion on pulmonary function tests in the adult population of Mid-Anatolia. Soz Praventivmed. 2004; 49(4):247–53. [PubMed: 15357526]
14. Corbo GM, Forastiere F, Agabiti N, Dell'Orco V, Pistelli R, Aebischer ML, et al. Effect of gas cooking on lung function in adolescents: modifying role of sex and immunoglobulin E. Thorax. 2001; 56(7):536–40. [PubMed: 11413352]

15. Jarvis D, Chinn S, Luczynska C, Burney P. Association of respiratory symptoms and lung function in young adults with use of domestic gas appliances. Lancet. 1996; 347(8999):426–31. [PubMed: 8618483]
16. Moran SE, Strachan DP, Johnston ID, Anderson HR. Effects of exposure to gas cooking in childhood and adulthood on respiratory symptoms, allergic sensitization and lung function in young British adults. Clin Exp Allergy. 1999; 29(8):1033–41. [PubMed: 10457105]
17. ATS. Standardization of Spirometry. Am J Respir Crit Care Med. 1995; 152:1107–36. [PubMed: 7663792]
18. SPSS. SPSS for Windows. Chicago: Apache Software Foundation; 2003.
19. Dezateux C, Stocks J. Lung development and early origins of childhood respiratory illness. Br Med Bull. 1997; 53(1):40–57. [PubMed: 9158283]
20. Tepper RS, Williams-Nkomo T, Martinez T, Kisling J, Coates C, Daggy J. Parental smoking and airway reactivity in healthy infants. Am J Respir Crit Care Med. 2005; 171(1):78–82. [PubMed: 15502114]
21. Gilliland FD, Berhane K, McConnell R, Gauderman WJ, Vora H, Rappaport EB, et al. Maternal smoking during pregnancy, environmental tobacco smoke exposure and childhood lung function. Thorax. 2000; 55(4):271–6. [PubMed: 10722765]
22. Hanrahan JP, Tager IB, Segal MR, Tosteson TD, Castile RG, Van Vunakis H, et al. The effect of maternal smoking during pregnancy on early infant lung function. Am Rev Respir Dis. 1992; 145(5):1129–35. [PubMed: 1586058]
23. Wang X, Wypij D, Gold DR, Speizer FE, Ware JH, Ferris BG Jr, et al. A longitudinal study of the effects of parental smoking on pulmonary function in children 6–18 years. Am J Respir Crit Care Med. 1994; 149(6):1420–5. [PubMed: 8004293]
24. Cook DG, Whincup PH, Papacosta O, Strachan DP, Jarvis MJ, Bryant A. Relation of passive smoking as assessed by salivary cotinine concentration and questionnaire to spirometric indices in children. Thorax. 1993; 48(1):14–20. [PubMed: 8434347]
25. Cuijpers CE, Swaen GM, Wesseling G, Sturmans F, Wouters EF. Adverse effects of the indoor environment on respiratory health in primary school children. Environ Res. 1995; 68(1):11–23. [PubMed: 7729382]
26. Cunningham J, Dockery DW, Speizer FE. Maternal smoking during pregnancy as a predictor of lung function in children. Am J Epidemiol. 1994; 139(12):1139–52. [PubMed: 8209873]
27. Nuhoglu C, Gurul M, Nuhoglu Y, Karatoprak N, Sonmez EO, Yavrucu S, et al. Effects of passive smoking on lung function in children. Pediatr Int. 2003; 45(4):426–8. [PubMed: 12911479]
28. Burchfiel CM, Higgins MW, Keller JB, Howatt WF, Butler WJ, Higgins IT. Passive smoking in childhood. Respiratory conditions and pulmonary function in Tecumseh, Michigan. Am Rev Respir Dis. 1986; 133(6):966–73. [PubMed: 3717768]
29. Venners SA, Wang B, Ni J, Jin Y, Yang J, Fang Z, et al. Indoor air pollution and respiratory health in urban and rural China. Int J Occup Environ Health. 2001; 7(3):173–81. [PubMed: 11513066]
30. Tredaniel J, Boffetta P, Saracci R, Hirsch A. Exposure to environmental tobacco smoke and adult non-neoplastic respiratory diseases. Eur Respir J. 1994; 7(1):173–85. [PubMed: 8143819]
31. Chen R, Tunstall-Pedoe H, Tavendale R. Environmental tobacco smoke and lung function in employees who never smoked: the Scottish MONICA study. Occup Environ Med. 2001; 58(9): 563–8. [PubMed: 11511742]
32. Eisner MD. Environmental tobacco smoke exposure and pulmonary function among adults in NHANES III: impact on the general population and adults with current asthma. Environ Health Perspect. 2002; 110(8):765–70. [PubMed: 12153756]
33. Carey IM, Cook DG, Strachan DP. The effects of environmental tobacco smoke exposure on lung function in a longitudinal study of British adults. Epidemiology. 1999; 10(3):319–26. [PubMed: 10230845]
34. Frette C, Barrett-Connor E, Clausen JL. Effect of active and passive smoking on ventilatory function in elderly men and women. Am J Epidemiol. 1996; 143(8):757–65. [PubMed: 8610685]
35. Jaakkola MS, Jaakkola JJ, Becklake MR, Ernst P. Passive smoking and evolution of lung function in young adults. An 8-year longitudinal study. J Clin Epidemiol. 1995; 48(3):317–27. [PubMed: 7897453]

36. EPA. Respiratory health effects of passive smoking: lung cancer and other disorders. Washington, DC: Office of Research and Development; 1992.
37. ALA. State of air 2002, fact sheet: COPD profiler. American Lung Association; 2002.
38. Anderson HR. Chronic lung disease in the Papua New Guinea Highlands. Thorax. 1979; 34(5): 647–53. [PubMed: 515985]
39. Dennis RJ, Maldonado D, Norman S, Baena E, Martinez G. Woodsmoke exposure and risk for obstructive airways disease among women. Chest. 1996; 109(1):115–9. [PubMed: 8549171]
40. Norboo T, Yahya M, Bruce NG, Heady JA, Ball KP. Domestic pollution and respiratory illness in a Himalayan village. Int J Epidemiol. 1991; 20(3):749–57. [PubMed: 1955261]
41. Perez-Padilla R, Regalado J, Vedal S, Pare P, Chapela R, Sansores R, et al. Exposure to biomass smoke and chronic airway disease in Mexican women. A case-control study. Am J Respir Crit Care Med. 1996; 154(3 Part 1):701–6. [PubMed: 8810608]
42. Behera D, Jindal SK, Malhotra HS. Ventilatory function in nonsmoking rural Indian women using different cooking fuels. Respiration. 1994; 61(2):89–92. [PubMed: 8008994]
43. Gold JA, Jagirdar J, Hay JG, Addrizzo-Harris DJ, Naidich DP, Rom WN. Hut lung. A domestically acquired particulate lung disease. Medicine (Baltimore). 2000; 79(5):310–7. [PubMed: 11039079]
44. Brutsaert TD, Parra EJ, Shriver MD, Gamboa A, Rivera-Ch M, Leon-Velarde F. Ancestry explains the blunted ventilatory response to sustained hypoxia and lower exercise ventilation of Quechua altitude natives. Am J Physiol Regul Integr Comp Physiol. 2005; 289(1):R225–34. [PubMed: 15802561]
45. Brutsaert TD, Spielvogel H, Soria R, Caceres E, Buzenet G, Haas JD. Effect of developmental and ancestral highaltitude exposure on VO(2)peak of Andean and European/North American natives. Am J Phys Anthropol. 1999; 110(4):435–55. [PubMed: 10564574]
46. Frisancho AR, Frisancho HG, Milotich M, Brutsaert T, Albalak R, Spielvogel H, et al. Developmental, genetic, and environmental components of aerobic capacity at high-altitude. Am J Phys Anthropol. 1995; 96(4):431–42. [PubMed: 7604895]
47. Greksa LP. Evidence for a genetic basis to the enhanced total lung capacity of Andean highlanders. Hum Biol. 1996; 68(1):119–29. [PubMed: 8907759]
48. Greksa LP, Spielvogel H, Caceres E, Paredes-Fernandez L. Lung function of young Aymara highlanders. Ann Hum Biol. 1987; 14(6):533–42. [PubMed: 3435039]
49. Zelikoff JT, Chen LC, Cohen MD, Fang K, Gordon T, Li Y, et al. Effects of inhaled ambient particulate matter on pulmonary antimicrobial immune defense. Inhal Toxicol. 2003; 15(2):131–50. [PubMed: 12528043]

Table 1

Baseline characteristics of children and adults in each cooking fuel category.

Cooking fuel category	Number	Gender M:F ratio	Age (years) mean (SD)	Height (cm) mean (SD)	ETS exposure # (%)
Children (7–15 years)					
LPG only	23	0.92	10.9 (2.6)	133.6 (11.4)	10 (43.5)
Primarily LPG	28	1.80	11.3 (2.2)	129.5 (11.4)	6 (21.4)
Primarily biomass	14	1.80	11.0 (2.0)	129.1 (10.3)	5 (35.7)
Biomass only	12	1.00 ($P = 0{:}59$) *	11.4 (2.7) ($P = 0{:}90$) †	131.7 (14.6) ($P = 0{:}59$) †	4 (33.3) ($P = 0{:}41$) *
Women (≥16 years)					
LPG only	24		34.3(13.2)	149.1 (6.8)	8 (33.3)
Primarily LPG	21		36.3 (12.6)	145.7 (4.9)	4 (19.0)
Primarily biomass	25		42.6 (17.6)	144.5 (4.6)	11 (44.0)
Biomass only	22		45.2 (18.7) ($P = 0{:}07$) †	142.7 (5.2) ($P = 0{:}002$) †	5 (22.7) ($P = 0{:}24$) *

* Comparison of frequencies using Pearson's χ^2 test.

† Comparison of means using ANOVA.

Table 2

Mean pulmonary function values for women and children in different cooking fuel categories.

	FVC mean (SD)	FEV_1 mean (SD)	FEV_1/FVC mean (SD)	$FEF_{25-75\%}$ mean (SD)
Children (7–15 years)				
LPG only	2.57 (0.69)	2.27 (0.68)	87.9 (9.7)	3.12 (1.16)
Primarily LPG	2.38 (0.55)	2.13 (0.54)	89.4 (5.4)	2.77 (0.95)
Primarily biomass	2.46 (0.57)	2.13 (0.49)	86.7 (5.6)	2.49 (0.75)
Biomass only	2.61 (0.84)	2.28 (0.65)	88.5 (6.0)	2.84 (0.68)
All children	2.48 (0.64)	2.20 (0.59)	88.3 (7.0)	2.83 (0.96)
Women (≥16 years)				
LPG only	3.58 (0.61)	3.02 (0.53)	84.4 (5.7)	3.55 (1.02)
Primarily LPG	3.42 (0.61)	2.81 (0.58)	81.8 (6.0)	3.02 (1.05)
Primarily biomass	3.16 (0.74)	2.65 (0.57)	84.4 (6.6)	3.00 (0.91)
Biomass only	2.99 (0.92)	2.53 (0.80)	84.9 (6.4)	2.90 (1.12)
All women	3.29 (0.76)	2.76 (0.64)	83.9 (6.2)	3.12 (1.04)

Table 3

Multivariate linear regression analyses comparing pulmonary function between children in different cooking fuel categories.

	FVC		FEV_1		FEV_1/FVC		$FEF_{25-75\%}$	
	β	95% CI	β	95% CI	β	95% CI	β	95% CI
Cooking fuel *								
Primarily LPG	−0.42†	(−0.70, −0.14)	−0.36†	(−0.67, −0.05)	0.66	(−6.21, 7.53)	−0.54	(−1.21, 0.12)
Primarily biomass	−0.51†	(−0.86, −0.16)	−0.54†	(−0.93, −0.16)	−3.55	(−12.04, 4.93)	−0.89†	(−1.72, −0.07)
Biomass only	−0.41†	(−0.76, −0.07)	−0.39†	(−0.78, −0.01)	0.45	(−8.03, 8.93)	−0.53	(−1.35, 0.29)
Gender *								
Males	−0.34†	(−0.64, −0.03)	−0.33	(−0.67, 0.01)	−0.37	(−7.84, 7.09)	−0.77†	(−1.49, −0.05)
Age (years)	0.13†	(0.08, 0.18)	0.14†	(0.08, 0.20)	1.37†	(0.11, 2.63)	−0.03	(−0.11, 0.16)
Height (cm)	0.02†	(0.01, 0.03)	0.02†	(0.00, 0.03)	−0.22	(−0.46, 0.14)	0.05†	(0.02, 0.08)
Tobacco smoke *								
Passive	−0.45†	(−0.77, −0.13)	−0.65†	(−1.00, −0.25)	−10.37†	(−18.22, −2.54)	−0.88†	(−1.68, −0.08)

* Reference categories are LPG only, females, and no tobacco smoke exposure.

† *P*-value <0.05.

Table 4

Multivariate linear regression analyses comparing pulmonary function between women in different cooking fuel categories.

	FVC		FEV_1		FEV_1/FVC		$FEF_{25-75\%}$	
	β	95% CI	β	95% CI	β	95% CI	β	95%CI
Cooking fuel*								
Primarily LPG	0.03	(−0.38, 0.43)	−0.11	(−0.42, 0.21)	−3.90	(−8.11, 0.30)	−0.54	(−1.13, 0.06)
Primarily biomass	0.10	(−0.34, 0.54)	0.03	(−0.32, 0.37)	−1.08	(−5.65, 3.49)	−0.16	(−0.80, 0.48)
Biomass only	0.02	(−0.40, 0.45)	0.05	(−0.28, 0.38)	2.25	(−2.18, 6.67)	−0.01	(−0.61, 0.64)
Age (years)	−0.02†	(−0.03, −0.02)	−0.02†	(−0.03, −0.02)	−0.14†	(−0.21, −0.05)	−0.04	(−0.05, 0.02)
Height (cm)	0.04†	(0.02, 0.06)	0.03†	(0.01, 0.05)	−0.10	(−0.34, 0.14)	0.03	(−0.01, 0.06)
Tobacco smoke*								
Passive	0.13	(−0.37, 0.62)	0.02	(−0.36, 0.41)	−2.05	(−7.16, 3.06)	−0.19	(−0.91, 0.53)

*Reference categories are LPG only and no tobacco smoke exposure.

†*P*-value <0.05.