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Respiratory Health and Indoor Air Pollution at High Elevation

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Abstract

In this research, the authors sought to provide experimental data on indoor air quality, and the resulting respiratory impact, for a high-elevation (4550 m), rural community in Ladakh, India. This community is of interest because the primarily nomadic residents burn biomass inside the home for heating and cooking. The concentrations of particulate matter (PM), endotoxin, and carbon monoxide were determined for 6 homes. Lung function data and induced sputum samples were collected for 9 female test-home subjects. In addition, lung function data were collected for 84 additional Ladakhi highlanders at this location. Sputum from 3 visiting scientists (sojourners) was collected and analyzed as well. The average PM concentration ranged from 2 mg/m^3 to 7 mg/m^3, with 85% of the sampled PM sized as respirable. The average endotoxin concentration ranged from 2.4 ng/m^3 to 19 ng/m^3, and average carbon monoxide levels ranged from 50 ppm to 120 ppm. Lung function values for the highlander population and the test-home subjects were equal to or greater than predicted, despite the highlanders' significant exposure to indoor pollutants. An induced sputum analysis revealed a significantly greater total inflammatory cell count ($M \pm SD$, 10^5 cell/mg) in the Ladakhi natives than in the sojourners (107.5 ± 75.2 vs 7.1 ± 8.1, p .01). Although the high levels of indoor pollutants did not correlate with significant decrements in lung function, the induced sputum analysis revealed marked airway inflammation dominated by macrophages and neutrophils. It appears that augmented lung mechanics of this high-altitude population are adaptive to reduce the work of breathing; thus, decrements in lung function go undetected because the true predicted values are greater than expected.

Keywords

biomass combustion; high-altitude population; indoor air quality; lung function

The World Health Organization estimates that 20% to 30% of all respiratory diseases are caused by ambient and indoor air pollution.1 Globally, indoor air contaminants are of

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concern because nearly 50% of the world's population burns biomass for domestic purposes such as cooking and heating.1,2 In developing nations, indoor exposure to biomass combustion byproducts has been associated with an increased incidence of respiratory disease as well as an increased rate of mortality.3–12 The World Bank has estimated that indoor air pollution is responsible for almost half the burden of disease in developing countries.2,13 In India alone, it is estimated that 400,000 to 550,000 premature deaths can be contributed to the use of biomass fuels.14 In Ladakh, Indian Himalaya, research has shown a prevalence of chronic cough, chronic phlegm, nonoccupational pneumoconiosis, and respiratory morbidity.15,16 We studied a unique population of nomadic people living at elevations greater than 4500 m (approximately 240 km southeast of Leh, Ladakh, India). These nomads rely on their animal herds for subsistence, and they use biomass (brush and animal dung) for fuel for cooking and heating. Most of the studied homes used a 50/50 mix of brush and dung (exceptions to this rule are noted). Because of this dependence on dung as a fuel source, we postulated that indoor endotoxin levels would be excessive. We further postulated that, as a result of indoor combustion, levels of particulate matter (PM) and carbon monoxide (CO) would be excessive. Finally, we expected that, with these high exposure levels, this population would have significant lung function decrements and marked airway inflammation.

To test these hypotheses, we did the following: (1) We measured the size distribution and concentration of PM present in domiciles where biomass is burned; (2) We determined the concentration of endotoxin in these environments; and (3) We measured the concentration of CO present. We collected these data in the form of both personal and area samples. We also collected induced sputum from female inhabitants of the studied domiciles and measured their lung function. Our secondary objectives included these: (1) determining if stove use improved indoor air quality and (2) determining the impact of the use of kerosene lanterns.

METHODS

Study Subjects and Sampling Conditions

We conducted this study in a high-altitude, agrarian community adjacent to Lake Tsomoriri in the upper Rupshu Valley approximately 240 km southeast of Leh, Ladakh, India. This area is free from all industrial pollution, and smoking tobacco seemed to be limited to men. We recruited subjects from the Village of Korzok (4550 m) and nomad camps (4550−4800 m) in the surrounding area. We surveyed subjects as to age, exposure to indoor air pollution, tobacco use, and living structure. We collected pulmonary function data and induced sputum samples for 9 test-home subjects (women, aged 18−69 years; mean age 41 years). We also measured pulmonary function in 84 additional Ladakhi highlanders (52 men, 32 women). All of these subjects lived in the Village of Korzok or the surrounding area. Three visiting scientists, herein referred to as sojourners (2 men, 1 woman; aged 19−45 years), all members of the research team, provided sputum samples for analysis. The study received approval from the Summa Health Care System, Human Subjects Committee, India and the Institutional Review Board at the University of California at Davis. Prior to the subjects' participation, we obtained informed consent in their native language to ensure that the design and risks of the study were clearly presented to all the subjects.

Working (eg, farming, herding) residents of this community spent approximately 5 hours per day exposed to bio-mass combustion byproducts. Exposure time was higher for those residents who did not routinely work outdoors during the day (some women, children, or elderly individuals), as fires were started for midday meals or warmth. Women were the predominant focus of this study because they were the most highly exposed during meal preparation. We assessed indoor air in 6 homes located within 3 miles of each other. The simple homes consisted of 1 room with a dirt floor; they averaged 25 m^2, with a ceiling height of approximately 1.7 m. Four of the homes (Homes 1, 3, 5, and 6) were portable tentlike structures, called yurts, made of a black, heavy, open-weave burlap material with large gaps at the door (Figure 1). One home (Home 4) consisted of a portable, tightly sealed canvas tent, whereas another home (Home 2) was a permanent stone structure with a wood door and plastic-covered window (Figure 2). Three of the homes (Homes 1, 3, and 6) had roof holes positioned directly over an open-pit fire to exhaust smoke; during inclement weather, this hole was covered and smoke remained within the domicile. Three of the homes (Homes 2, 4, and 5) had an open-front metal stove with an attached chimney that extended through the roof of the dwelling. Kerosene lamps were present in 2 homes (Homes 1 and 2) and were used in the evening and early morning.

We performed indoor sampling during meal preparation. The family cook knelt or sat on the ground. We obtained personal samples from the woman performing the cooking, and used a strap to position a sampling pump, cassette, and filter as close to the breathing zone as possible. We obtained area samples approximately 0.76 m away from the fire or stove, and 0.6 m above the ground, positioning the height of the inlet close to breathing height. Temperatures at this high-altitude location ranged from −12°C at night to 21°C during the day; the relative humidity averaged 20%.

Particulate Matter

We measured the size distribution of the PM in Home 1 by using a Sierra 210 cascade impactor (Graseby-Andersen, Smyrna, GA) with aluminum substrates and a glass-fiber final filter. We did not deem particle bounce to be an issue because airborne indoor PM was predominantly composed of smoke. Because of the lack of electricity and the limitations in generator movement caused by the terrain, we could not obtain impactor samples in other homes. We concurrently collected personal samples, area samples, or both with 37-mm sampling cassettes loaded with 1-μm-pore polytetrafluoroethylene filters attached to SKC personal sampling pumps (SKC, Eighty Four, PA). We followed sampling method ASTM D5955 for Environmental Tobacco Smoke (respirable particles) from the American Society for Testing and Materials (ASTM), but we did not use a cyclone to collect larger particles. We followed all calibration procedures recommended by the ASTM method. In all other residences (Homes 2−6), we collected between 1 and 3 samples with 37-mm sampling cassettes as just described. Sampling time ranged from 45 minutes to 1 hour, and we varied it with inlet concentration to ensure that we collected sufficient mass for accurate gravimetric analysis. In addition, we took 2 outdoor samples on closed-face 37-mm sampling cassettes to determine ambient PM levels. Sample time was 2 hours at a flow rate of 2 LPM. We took outdoor samples at a location absent of combustion sources. We carried field blanks for all samples.

We weighed the sampling filters, impactor substrates, and blanks in the United States before we left and after we returned by using a Mettler MT5 balance (Mettler-Toledo, Inc, Switzerland). We desiccated and acclimated all filters before each weighing. We developed a cumulative size distribution by mass for the impactor runs, from which we determined mass median aerodynamic diameter (MMAD) and geometric standard deviation (GSD). We calculated particle concentration by using polytetrafluoroethylene filter weights and sampling times, as well as total impactor substrate and filter weights and sampling times. We calculated 8- and 24-hour time-weighted averages (TWAs) for comparison with US ambient and occupational standards for PM. We assumed a 5-hour indoor exposure time, with background (outdoor) concentrations used for the remaining hours.

Carbon Monoxide

We monitored CO by using a passive color dosimeter tube (SKC, Eighty Four, PA) that provided a TWA. We collected 4 samples (2 runs) in Homes 1 and 2, and we collected 2 samples in Homes 3 through 6. We positioned one sample tube with the area sampling equipment, and we positioned the other tube to take a personal sample within the cook's breathing zone. We took 2 outdoor samples to determine ambient CO levels.

Endotoxin

Sampling followed the standard protocol provided by the laboratory to analyze the samples (P & K Microbiology Services, Inc, Cherry Hill, NJ).17 We sampled endotoxin by using a 3-piece 37-mm sampling cassette preloaded with 0.45- m polycarbonate filters provided by the analytical laboratory. We obtained 2 sets of area samples from both Homes 1 and 2. We used sampling times that ranged from 45 minutes to 1 hour to ensure that filters would not clog as the result of high levels of indoor PM contaminants. We took a 2-hour outdoor sample to determine ambient endotoxin levels. We analyzed the samples, along with a blank, by using the Limulus amebocyte lysate assay kinetic chromogenic method.17 The detection limit for this analytical method was 0.0050 endotoxin units, or 0.0003 ng.

Pulmonary Function Measurements

We measured pulmonary function by using a portable spirometer (MicroLoop, Micro Medical, UK) on subjects who had been resting for at least 15 minutes. This fixed-orifice spirometer utilizes turbine technology and has an accuracy of 3%. Previous studies have shown that, with turbine-based spirometers, the lung function measures are not affected by either climate or altitude, unlike other spirometers that use different technologies.18,19 A trained interpreter explained the instructions for performing the forced vital capacity to subjects in their native language. Subjects wore nose clips and performed maneuvers in the seated position. To obtain at least 2 reproducible results, we had subjects perform between 3 and 6 forced expiratory maneuvers. In addition to measured values of lung volume and flows, the spirometer provided data on predicted values that were based on age, height, race (Caucasian or non-Caucasian), and sex, referenced to normal values published by the European Coal and Steel Community.20

Individuals from non-Caucasian races tend to have smaller lung volumes than do members of the Caucasian race for the same height and age. We adjusted for this in the MicroLoop

spirometer by multiplying final values by an adjustment factor of 0.9. In addition, we compared our data with those from a more recent study that provided spirometric reference values for Caucasians, African Americans, and Mexican Americans who were aged between 8 and 80 years; we also compared our values with available values for Asians.21–23

Induced Sputum Collection and Processing

We based our procedure for sputum induction and processing on previously described methods.24 The induction procedure involved 3 separate 7-minute inhalation periods of 3%, 4%, and 5% hypertonic saline, which we administered after the baseline spirometry. At the end of each 7-minute inhalation, the subjects performed a 3-step cleansing procedure prior to the cough attempt. We then instructed each subject to perform a “chesty type” of cough without clearing the back of the throat, and then to expectorate the sample into a sterile specimen cup that was placed on ice throughout the procedure. We began the sample processing immediately after we collected the sputum.

We manually selected and weighed mucus plugs, incubated (15 minutes at room temperature) them in 0.1% dithiothreitol (Calbiochem Corp, San Diego, CA), washed them with Dulbecco's phosphate-buffered saline, and gravity filtered them through a 48 micron pore mesh filter (BBSH Thompson, Scarborough, Ontario). We performed total cell counts using a Neubauer Hemocytometer. We neither counted visually identifiable squamous epithelial cells nor included them in the total cell count. We determined cell viability by using Trypan Blue exclusion staining. We performed a differential leukocyte analysis of nonsquamous cells (Diff Quik stained) on a minimum of 300 cells, provided that squamous epithelial cells were less than 40% of the total cells. We express differential cell counts (lymphocytes, neutrophils, eosinophils, monocytes, and macrophages) as a percentage of total nonsquamous nucleated cells.

Statistical Analysis

We analyzed the spirometry data by using a Kruskal-Wallis analysis of variance (nonparametric) with post hoc nonpara-metric comparisons made with Dunn's test. We analyzed the data for the percentage of predicted lung function by using the Wilcoxon test to determine whether the group values differed significantly from the theoretical mean of 100%. We analyzed the sputum and air sampling data by using an un-paired two-sided *t* test. We set the alpha level for significance at $\alpha = 0.05$ for all comparisons. We performed our calculations by using SAS version 8.2 and Excel 2003. We present the data as mean ± *SD*, unless otherwise noted.

Comparison with Literature and Standards

We compared all data (exposure, spirometry, and sputum) with data available in the literature. In addition, we compared the sampling results with US occupational and ambient air-quality standards, to put into perspective the contaminant levels to which this population is routinely exposed. Occupational standards, set by Occupational Safety and Health Administration (OSHA) and the National Institute for Occupational Safety and Health (NIOSH), are applied in the workplace and are established to protect healthy, work-aged individuals. National Ambient Air Quality Standards (NAAQS), set by the US

Environmental Protection Agency (USEPA), are applied indiscriminately across the entire nation to protect the health of the general population (including asthmatics, children, and the elderly).

RESULTS

Particulate Matter

We measured the size distribution of PM in Home 1. When a lantern was used in Home 1, the aerosol had an MMAD of 0.15 m (GSD = 35) and a concentration of 5.33 mg/m^3 (based on summation of mass on all impactor substrates). Without the lantern, the aerosol had a larger MMAD of 0.4 m (GSD = 9) and a somewhat lower impactor-based concentration of 3.66 mg/m^3. Given the high GSDs, we see that the distribution is obviously multimodal. We determined 85% of the sampled PM to be respirable, that is, reaching or capable of reaching the gas-exchange region of the lungs, according to American Conference of Governmental Industrial Hygienists (ACGIH) criteria.25 Overall average household concentrations for PM ranged from 2 to 7 mg/m^3. Personal samples tended to show a 1.5-fold higher concentration than did area samples taken concurrently (Table 1). Figures 3 and 4 show the TWA PM levels measured during biomass burning in homes, with and without kerosene lamp use (Homes 1 and 2). When a kerosene lamp was used, PM levels increased for both personal and area samples.

Carbon Monoxide

CO levels ranged from 50 to 120 ppm, with maximum levels occurring while kerosene lamps were used (Table 2). Figure 5 shows CO levels measured in Home 1 during biomass burning alone versus those measured during biomass burning and kerosene lamp use. Stove use did not seem to have any effect on CO level (p .2). Personal samples provided the same result as area samples taken concurrently.

Endotoxin

Average endotoxin levels measured in Homes 1 and 2 were 19 and 2.4 ng/m^3, respectively. Biomass content (% brush vs % animal dung) appeared to govern the level of endotoxin present. The residents of Home 1 kept livestock and dried animal dung within 61 cm (2 ft) of their home, and thus burned more animal dung than did other homes. Home 2 was further removed from livestock and thus used dung sparsely. Indoor endotoxin levels during biomass combustion were higher for both homes than for those observed for outdoor samples (0.07 ng/m^3).

Pulmonary Function

Pulmonary function data, expressed as the percentage predicted for the test-home subjects and overall Ladakhi highlander population, are presented in Figure 6. We predicted lung function on the basis of ethnic background, sex, and age of the individual.20 The values for forced vital capacity (FVC), forced expiratory volume at 1 second (FEV_1), FEV_1 to FVC ratio (FEV_1:FVC), and maximum mid-expira-tory flow (MMEF) for the Ladakhi highlander population (women and men combined) were significantly greater ($p < .05$) than were the predicted values for non-Caucasians.20 The test-home subjects showed significantly lower

($p < .05$) predicted values than the general highland population for FEV_1:FVC and MMEF. However, the actual mean values for FVC, FEV_1, FEV_1:FVC, and MMEF were not significantly different from the theoretical mean (100% predicted for age, sex, size, and race).

Airway Inflammation

An induced sputum analysis revealed a significant airway inflammation in all of our test-home subjects. The total inflammatory cell count ($M \pm SD$, 10^5 cell/mg) was 14-fold greater in the Ladakhi natives than in the sojourner scientists (107.5 ± 75.2 vs 7.1 ± 8.1, $p < .01$). The differential analysis showed a marked neutrophilic inflammation in the test-home subjects, with 34.1 12.1% neutrophils, compared with 15.3 6.4% neutrophils in the sojourners ($p < .02$). Furthermore, the absolute cells counts (total cell count %, $M \pm SD$,10^5 cell/mg) for both macrophages and neutrophils were significantly greater ($p < .02$) in the test-home subjects (61.6 ± 34.4 and 45.6 ± 41.8, respectively) than in the sojourners (6.2 ± 7.6 and 0.7 ± 0.6, respectively). The macrophages from the test-home subjects tended to be highly vacuolated, with many of the cells actively engaged in the phagocytosis of particles. Furthermore, we found large numbers of variously sized free particles in the sputum of the test-home subjects.

DISCUSSION

Particulate Matter

During biomass combustion, PM is released both as smoke and ash; this is the reason for the multimodal size distribution that we discussed earlier. Impactor results show that 85% of the indoor aerosol was in the respirable range. Standards set by OSHA limit 8-hour respirable PM (nuisance dust) exposure to 5 mg/m^3, as determined by personal sampling.26 All homes fell below this exposure limit during all sampling runs. The 24-hour NAAQS for PM_{10}, which is PM with a diameter of 10 m or smaller, is 150 $\mu g/m^3$, according to the USEPA.27 All homes fell well above the USEPA's NAAQS for PM_{10}. Figures 3 and 4 show the PM 24-hour and 8-hour TWAs for each home, and they compare these TWAs with the aforementioned standards. Levels of ambient respirable PM that are much lower than the indoor levels we found in this study have been associated with asthma exacerbation and bronchitis, as well as an increase in mortality for those with preexisting respiratory or cardiovascular disease.28–30

Contrary to our expectations, the use of a stove made no statistically significant difference in the PM levels ($p = .4$). Although this may have been due to the small sample size, other likely explanations for this unexpected finding include faulty stove operation, poor stove design, or poor ventilation in homes with stoves. Indeed, all stoves were operated with their doors open so fuel could be fed to the fire; smoke visibly escaped into the homes through these openings. Furthermore, homes with stoves appeared to be more airtight than homes without stoves. Although we did not determine air-exchange rates, structural differences were obvious. Homes without stoves had large holes cut into the roof to allow smoke to escape and doors that were merely tent fabric pulled together with rope. Homes with stoves had stovepipes running through sealed holes in the roof and doors that closed or zipped

tightly and effectively sealed in the smoke. Home 4, the tightly sealed tent, had the highest indoor PM concentrations without the use of a lantern.

Numerous studies at varying elevations have investigated indoor PM concentrations as a result of combustion of biomass fuels. Table 3 summarizes the results of several studies,31–34 including the present study. (Note than only one other study has investigated the levels of indoor PM caused by biomass combustion at high altitude, and that study did not have a respirable PM measure for comparison.3) Although the particulate concentrations for each of these studies were determined with differing cut sizes (ie, respirable <4 μm, $PM_{2.5}$, or PM_{10}), our values fell within the published ranges for all elevations. In looking at these data, we find that altitude does not appear to affect indoor PM levels.

Carbon Monoxide

CO is a colorless, odorless gas that is emitted during the incomplete combustion of carbonaceous materials, such as biomass. Human exposure to CO results in bloodstream oxygen replacement and increased carboxyhaemoglobin levels. Even low levels of CO inhalation can result in a significant decrease in oxygen delivered to the body tissues.35 We calculated 8-hour TWAs for comparison with occupational CO standards. Again, we assumed a 5-hour daily exposure time, with background (outdoor) concentrations used for the remaining 3 hours. The 8-hour TWA threshold set by NIOSH for CO is 35 ppm,3 and the 8-hour permissible exposure limit set by OSHA is 50 ppm.26,36 As one can see in Figure 5, the 8-hour CO TWAs for 4 of the 6 homes fell at or above the threshold established by NIOSH, and 1 of the 6 sampled homes fell above the limit set by OSHA. The USEPA's 1-hour NAAQS for CO is 35 ppm and its 8-hour NAAQS is 9 ppm.27 All 6 homes fell well above both of these standards. CO can accumulate in the blood as a result of chronic exposure to low concentrations (10−30 ppm), and clearance of CO from the blood is quite slow (3- to 5-hour half-life). Whereas healthy individuals typically adapt to the decrease in blood oxygen level by vasodilation, patients with cardiovascular disease are unable to substantially increase coronary flow. As a result, at the CO levels found in this study, myocardial hypoxia becomes a real possibility for individuals with cardiovascular compromise.28

Few studies have investigated CO that is generated from noncoal biomass combustion in conditions that are similar to those of the current study (rural, primitive homes with primitive cooking means). One similar study showed CO concentrations ranging from 30 to 50 ppm in both well-ventilated and nonventilated Guatemalan kitchens during the combustion of unprocessed biomass fuels.37 This same study also found that, despite similar indoor CO concentrations, carboxyhaemoglobin levels were significantly higher in the blood of women exposed to biomass combustion at higher elevations (250 m vs 1350 m). Indeed, at the higher altitudes the oxyhemoglobin dissociation curve is shifted to the left and thus has a greater affinity for both oxygen and CO. Furthermore, binding of CO to hemoglobin will also left-shift the curve, thus further increasing the binding affinity for both molecules. In wood-burning homes in Costa Rica, Park and Lee33 found maximum 1-minute CO levels during cooking to range from 2.1 ppm to 62.6 ppm. The CO concentrations reported in these 2 studies were generally lower than those found in the

current study. As one can see in Table 2, average data from the current study ranged from 50 ppm to 120 ppm CO during cooking.

Endotoxin

Endotoxins are a component of the outer membrane of Gram-negative bacteria that contain lipopolysaccharide and other naturally occurring compounds. During the combustion of biomass, particularly animal dung, endotoxins are released constituents of incompletely combusted PM. Exposure to airborne endotoxin, in both residential and occupational settings, can cause acute and chronic airway inflammation, decreased lung function, and an increase in asthma severity.38–42 There are currently no established threshold values or standards for endotoxin, but studies suggest that disease is related to endotoxin exposure level. On the basis of such studies, guidelines for “no effect” of environmental endotoxin have been developed.43 At 10 ng/m^3, airway inflammation may occur. Chronic endotoxin airway inflammation has been shown to lead to a decline in lung function over several years.

As we discussed earlier, Home 1's average endotoxin level is high enough to cause airway inflammation. This home appeared to burn more animal dung than brush, which was likely the cause of the increased endotoxin levels (compared with Home 2). Over several years, exposure to this endotoxin level is likely to cause decreased lung function in the residents of this home, and it is likely a major cause for lung irritation reflected by increased levels of macrophages and neutrophils in sputum. It should be noted that, as a result of the agrarian nature of this community, endotoxin released during dung combustion is merely one exposure source for this population. We found no data in the current literature regarding exposure to endotoxin from biomass combustion.

Pulmonary Function

We found well-preserved pulmonary function in both our test-home subjects and the general Lahdaki highlander population, despite significant indoor levels of particulates, endotoxin, and CO. Our findings differ from other studies investigating pulmonary function and indoor biomass combustion exposure.44–46 Pandey et al44 showed that both FEV_1 and FVC decreased with the duration of biomass pollutant exposure in Nepalese villagers residing at an altitude of 1300 m. However, the observed decrease in lung function was confounded by smoking, as smokers showed a more pronounced decrease than nonsmokers. In another study, Turkish villagers exposed to long-term biomass combustion suffered severe obstruction with a mean FEV_1 of 41.8% predicted and a FVC of 51% predicted.45 Behera et al46 reported decreased FVC values but normal FEV_1 and FEV_1:FVC values in lowland Indian villagers who were routinely exposed to indoor biomass pollutants.

CONCLUSIONS

We studied a population that resided at 4550 m, which is an altitude over 3 times higher than that of other studies in the literature. Indeed, Korzok is one of the highest permanently inhabited villages in the world. We determined that indoor air pollution that is attributable to the combustion of biomass is significant in the homes of this area. Our findings of greater than predicted lung function are similar to thse reported previously for Lahdaki highlanders.

47,48 Wood et al47 suggested that the augmented lung mechanics of this high-altitude population is adaptive to reduce the work of breathing. Apte and Rao48 further suggested an inherited adaptive mechanism resulting in greater lung elastic recoil and larger cross section of the small airways. It is possible that decrements in lung function go undetected in this unique population because the true predicted values are greater than expected.

Our study clearly demonstrates airway inflammation suggestive of chronic bronchitis in our test-home subjects. Phagocytosis by activated macrophages is an important mechanism for airspace clearance of particulates and bacteria; it is an active function and requires significant energy. Hypoxia has been show to have an inhibitory effect on the phagocytic activity of alveolar macrophages.49,50 Therefore, at this altitude, it is possible that the phagocytic activity of macrophages and neutrophils are reduced. This may partially contribute to the apparently high occurrence of respiratory infections and chronic bronchitis in Ladakhi highlanders. Thus, between the elevated exposure levels to numerous contaminants, the evidence in the literature of serious health effects from these contaminants, and the documented disease in the Ladakhi population, there is a need for additional research leading to healthier living conditions for this population, despite their preserved respiratory mechanics.

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Fig. 1.
Tent home (yurt) as described for Homes 1, 3, 5 and 6.

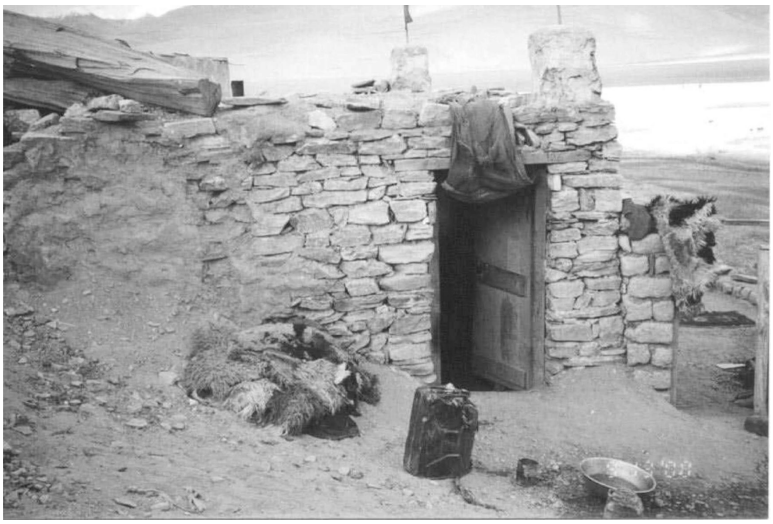

Fig. 2.
Stone home as described for Home 2.

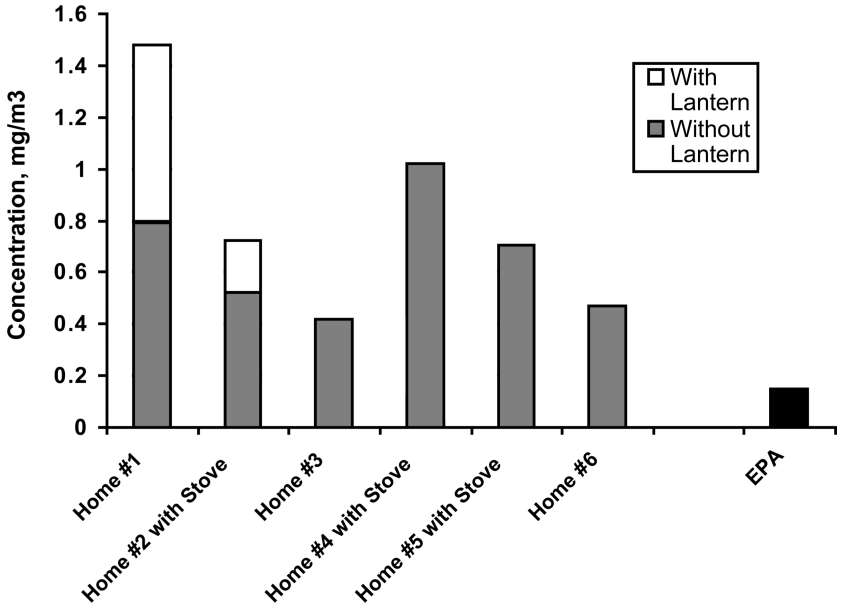

Fig. 3.

PM Levels (24-hour TWA) versus US Environmental Protection Agency (EPA) ambient standard for PM_{10}.

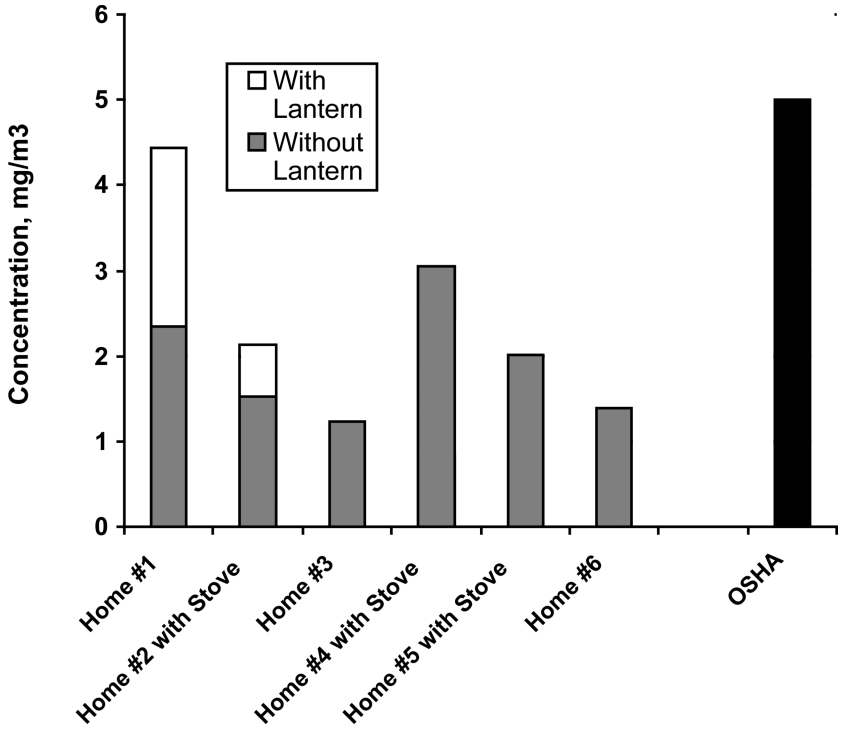

Fig. 4.

PM Levels (8-hour TWA) versus US Occupational Safety and Health Administration (OSHA) standard.

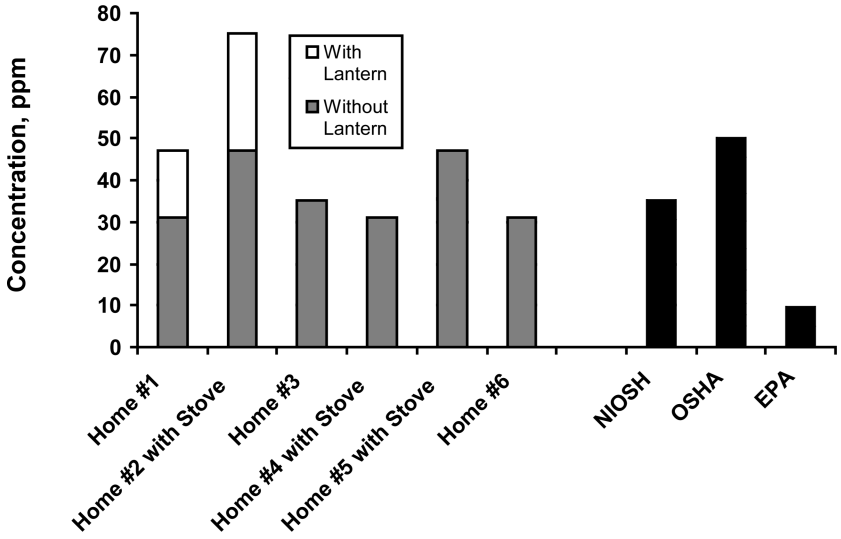

Fig. 5.

CO Levels (8-hour TWA) versus US Occupational and Ambient Standards, National Institute for Occupational Safety and Health (NIOSH), OSHA, and EPA.

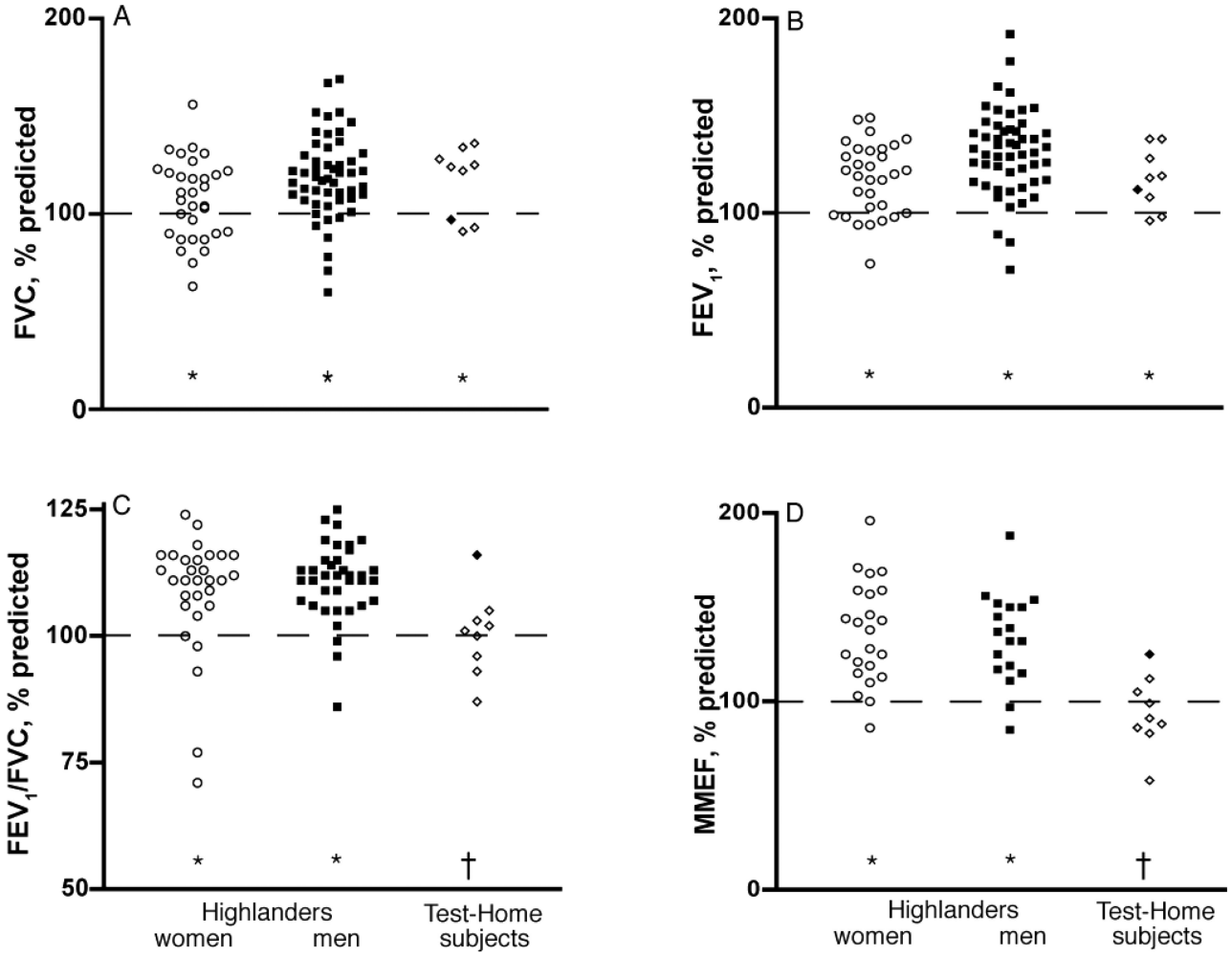

Fig. 6.

Lung function values for all subject groups expressed as percentage of predicted values. (A) shows % predicted forced vital capacity (FVC); (B) shows % predicted forced expiratory volume in one second (FEV_1); (C) shows % predicted FEV_1:FVC ratio; (D) shows % predicted maximum mid-expiratory flow (MMEF). * indicates that the actual mean is significantly different ($p < 0.05$) from the theoretical mean (100). † indicates that the group mean is significantly different than the group values for the general Ladahki Highlander population. Open symbols are women, and closed symbols are men.

Table 1

–Household Sampling Results for Particulate Matter

	Sampling pump data (mg/m^3)		
Home	**Avg. Conc.***	**8-h TWA**	**24-h TWA**
1: No lantern	3.75†	2.35†	0.79†
	2.81‡	1.76‡	0.59‡
1: Lantern	7.08 ± 3.05†	4.43 ± 1.9†	1.48 ± 0.64†
	5.95 ± 1.44‡	3.73 ± 0.9‡	1.25 ± 0.3‡
2: No lantern	2.44†	1.53†	0.52†
2: Lantern	3.42 ± 0.44‡	2.14 ± 0.27‡	0.72 ± 0.09‡
3	1.95†	1.23†	0.42†
4	4.9†	3.05†	1.02†
5	3.3†	2.01†	0.704†
6	2.2†	1.38†	0.47†
Outside	0.01 ± 0.003	. . .	. . .

*This is the average concentration during meal preparation.

†Personal sample.

‡Area sample.

Table 2

–Household Sampling Results for Carbon Monoxide

Home	Avg. Conc.* (ppm)	8-h TWA (ppm)
1: No lantern	50 ± 0†	31 ± 0
1: Lantern	75 ± 0†	47 ± 0
2: No lantern	75 ± 0†	47 ± 0
2: Lantern	120 ± 0†	75 ± 0
3	50 ± 0†	35 ± 0
4	50 ± 0†	31 ± 0
5	75	47
6	50	31
Outside	Below Detection‡	. . .

*This is the average concentration during meal preparation.

†Duplicates gave the same reading as the original sample.

‡The detection limit is 10 ppm.

Table 3

–Summary of PM Concentrations for Various Studies Evaluating Biomass Combustion

Reference/ Sample Location	Altitude (m)	Biomass type/ stove	PM concentration (mg/m^3)		
			Respirable (<4 μm) PM	$PM_{2.5}$	PM_{10}
Albalak et al 1999/Bolivia[3]	4100	Not defined	. . .	. . .	1.8
Balakrishnan et al 2002/Southern India31	Sea level	Wood and crop residues	0.5−2.0	. . .	. . .
Balakrishnan et al 2004/Andhra Pradesh, India32	Sea level	Wood, dung, crop residues	0.7	. . .	. . .
Ezzati et al 2000/Kenya5	1800	Wood or charcoal	. . .	N/A	2.8−4.9
Park and Lee 2003/Costa Rica33	Sea level	Wood/stove	. . .	0.3−8.2	0.5−18.9
Perez-Padilla et al 1999/Mexico34	Unknown	Wood	. . .	. . .	>1.0
Present Study	4550	Brush and dung	2.0−7.0	. . .	. . .