Household Stove Improvement and Risk of Lung Cancer in Xuanwei, China

Qing Lan, Robert S. Chapman, Dina M. Schreinemachers, Linwei Tian, Xingzhou He

Background: **Lung cancer rates in rural Xuanwei County, Yunnan Province, are among the highest in China. Residents traditionally burned "smoky" coal in unvented indoor firepits that generated very high levels of air pollution. Since the 1970s, most residents have changed from firepits to stoves with chimneys. This study assessed whether lung cancer incidence decreased after this stove improvement.** ***Methods:*** **A cohort of 21 232 farmers, born from 1917 through 1951, was followed retrospectively from 1976 through 1992. All subjects were users of smoky coal who had been born into homes with unvented firepits. During their lifetime, 17 184 subjects (80.9%) changed permanently to stoves with chimneys. A hospital record search detected 1384 cases of lung cancer (6.5%) during follow-up. Associations of stove improvement with lung cancer incidence were analyzed with product-limit plots and multivariable Cox models. In 1995, indoor concentrations of airborne particles and benzo[*a*]pyrene were compared in Xuanwei homes during smoky coal burning in stoves with chimneys and in unvented stoves or firepits.** ***Results:*** **A long-term reduction in lung cancer incidence was noted after stove improvement. In Cox models, risk ratios (RRs) for lung cancer after stove improvement were 0.59 (95% confidence interval [CI] = 0.49 to 0.71) in men and 0.54 (95% CI = 0.44 to 0.65) in women (for both, *P*<.001). Incidence reduction became unequivocal about 10 years after stove improvement. Levels of indoor air pollution during burning with chimneys were less than 35% of levels during unvented burning.** ***Conclusion:*** **Changing from unvented to vented stoves appears to benefit the health of people in China and may do so in other developing countries as well. [J Natl Cancer Inst 2002;94:826–35]**

The unvented household burning of coal and biomass fuel is very common in developing countries, particularly in rural areas. This type of burning usually generates very high levels of indoor air pollution and is an important risk factor for malignant and nonmalignant respiratory illness *(1–6)*. Previous studies have shown that stove improvements, such as the installation of chimneys, can substantially reduce indoor air pollution *(7,8)*. Thus, it is reasonable to hypothesize that stove improvements would reduce exposure to indoor air pollution and thereby reduce the risk of respiratory illness of people in developing countries. To the best of our knowledge, no previous studies have documented such a reduction in risk after stove improvement.

We tested this hypothesis with a retrospective cohort study in rural Xuanwei County, Yunnan Province, China. In this county, lung cancer mortality rates are among the highest in China *(9)*. From 1973 through 1979, annualized age-adjusted rates of lung cancer mortality in Xuanwei were 27.7 and 25.3 per 100 000 in men and women, respectively *(3)*. From 1973 through 1975, the annualized age-adjusted lung cancer mortality rate was considerably lower in China (6.8 in men and 3.2 in women) and was lower still in Yunnan as a whole (4.3 in men and 1.5 in women). In Xuanwei, more than 90% of residents are farmers with little or no exposure to industrial or automotive air pollution, and residential stability is very high. Most men, but very few women, smoke tobacco. Nearly all women, and some men, cook food on the household stove.

For household cooking and heating, Xuanwei residents have traditionally burned "smoky coal," "smokeless coal," or wood in unvented indoor firepits. (Smoky coal and smokeless coal are general descriptive terms used throughout China for bituminous and anthracite coal, respectively.) Burning smoky coal in a firepit generates very high indoor concentrations of airborne particulate matter (sometimes exceeding 20 mg/m^3), benzo[*a*]-pyrene, and other organic compounds *(3,10)*. Epidemiologic studies have shown that burning smoky coal is an important risk factor for lung cancer in Xuanwei *(3,11)*. Toxicologic studies have shown that combustion products from Xuanwei smoky coal are more tumorigenic and mutagenic than combustion products from smokeless coal and wood products *(3,12,13)*.

In recent decades, most Xuanwei residents have changed from firepits to stoves with chimneys. Residents have generally changed stoves at their own expense, although in 1976 the government offered a small one-time subsidy of 10 yuan (about $5 U.S. at that time) to assist with stove improvement. Anecdotal evidence strongly suggests that some residents did not change stoves until they had already developed respiratory symptoms; sometimes they changed on a physician's advice.

The cohort study, described below, tested whether the incidence rate of lung cancer decreased in subjects who changed from unvented firepits to stoves with chimneys, compared with that in subjects who did not change.

Methods

Cohort Study Design and Data Collection

In 1992, local administrative records were searched to identify the cohort of all farmers born from 1917 through 1951 and living in Xuanwei's three central communes (hereafter desig-

Affiliations of authors: Q. Lan, Institute of Environmental Health and Engineering, Chinese Academy of Preventive Medicine, Beijing, China, National Cancer Institute, National Institutes of Health, Bethesda, MD; R. S. Chapman, National Center for Environmental Assessment, U.S. Environmental Protection Agency, Research Triangle Park, NC; D. M. Schreinemachers, National Health and Environmental Effects Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park; L. Tian, X. He, Institute of Environmental Health and Engineering, Chinese Academy of Preventive Medicine.

Correspondence to: Robert S. Chapman, M.D., M.P.H., MD-52, National Center for Environmental Assessment, U.S. Environmental Protection Agency, Research Triangle Park, NC 27711 (e-mail: chapman.robert@epa.gov).

See "Notes" following "References."

nated the study area) as of January 1, 1976. The study area encompasses about 175 square miles. Lung cancer mortality in this area is higher than elsewhere in Xuanwei, and most area residents use smoky coal *(3)*. In all, 31 364 farmers were identified. Records of six hospitals were searched to identify all diagnoses of lung cancer in these farmers from 1976 through 1992. Four hospitals were in Xuanwei. One of these, the Xuanwei County Hospital, accounts for more than 90% of the county's hospitalizations. Records of the Qujing District Hospital and the Yunnan Province Hospital were also searched. (In China, a district comprises several counties and a province comprises several districts.) All subjects lived within 10–12 miles of a catchment hospital. The age of each patient in years at lung cancer diagnosis and clinical methods of diagnosis were recorded. Dates and causes of all deaths in the cohort from 1976 through 1992 were also recorded from death certificates.

From 1976 through 1992, 1215 (3.9%) of the original 31 364 farmers moved out of the study area and were not included in this analysis. Throughout 1992, trained interviewers administered a standardized questionnaire for all 30 149 farmers who had not moved. Subjects were interviewed directly when feasible; otherwise, surrogate respondents were interviewed. The questionnaire ascertained life history of household stove and fuel use, smoking, cooking food, time spent indoors and outdoors, education, residence locations, and illnesses in subjects and their relatives. This study was approved by the Institutional Review Board of the Chinese Academy of Preventive Medicine. Written informed consent was obtained from all literate questionnaire respondents. Study goals and procedures were thoroughly explained orally to each eligible person who was illiterate, in the presence of a literate relative. If the illiterate person gave oral consent, then the literate relative signed the consent form for that person. The questionnaire was administered to illiterate persons who consented in this fashion.

Cohort Data Statistical Analysis

Analysis was restricted to lifelong smoky coal users, born into households with unvented firepits, so that 6560 subjects were excluded. The 2196 subjects who changed from firepits to portable stoves were also excluded. There were 156 additional exclusions for missing data and questionnaire coding errors. Five lung cancer diagnoses were excluded because they lacked chest x-rays. Thus, 21 232 subjects (11 168 men and 10 064 women) were included in the analysis. During their lifetime, 17 184 subjects (80.9%) changed permanently to stoves with chimneys. The hospital record search detected 1384 diagnoses of lung cancer (6.5%) during follow-up.

For each subject, the age at entry into follow-up was calculated as the number of days from the subject's birth date to January 1, 1976, divided by 365.25. After entry, subjects could exit from follow-up in one of the following three ways (with method of calculating age at exit shown in parentheses): 1) being diagnosed with lung cancer (integer age at diagnosis + 0.5), 2) dying without lung cancer before the study ended in 1992 (number of days from birth date to death date, divided by 365.25), or 3) remaining alive and free of lung cancer at the time of interview in 1992 (number of days from birth date to interview date, divided by 365.25). Integer ages at entry into and exit from follow-up ranged from 24 to 58 years and from 25 to 75 years, respectively.

Firepits and stoves without chimneys do not vent smoke outdoors. Preliminary analysis showed no difference in lung cancer incidence between subjects who always used firepits and those who changed from firepits to stoves without chimneys. Thus, these two groups were combined into a single reference group without stove improvement. In this article, the term stove improvement is therefore synonymous with changing permanently from firepit to stove with chimney.

There were 313 579 person-years during follow-up (124 363 before stove improvement and 189 216 after it). Among the person-years before improvement, 47 053 (37.8%) were in subjects who never had improvement, and 77 310 (62.2%) were before stove improvement in those with stove improvement. Age-adjusted lung cancer incidences per 100 000 person-years were calculated before and after stove improvement. Survival curves, with lung cancer incidence as the outcome variable, were plotted separately by age for men and women with and without stove improvement (Statistical Analysis System [SAS] LIFETEST Procedure; SAS Institute Inc., Cary, NC). The product-limit method was used to calculate survival function estimates. The LIFETEST Procedure tests for homogeneity of curves in different strata with Wilcoxon, log-rank, and likelihood-ratio χ^2 statistics.

Sex-specific multivariable Cox models were constructed (SAS PHREG procedure). The time variable (time axis) was age, starting with age as of January 1, 1976, and ending with age at exit from follow-up. The outcome event was diagnosis of lung cancer (incident lung cancer). For each sex, a preliminary model included a zero-one indicator (dummy) variable for stove improvement. The goodness of fit of these preliminary models was tested by the inclusion of an additional variable for the interaction of stove improvement with the time variable, age. This interaction was highly statistically significant at $\alpha = .05$ (P<.001 for both sexes). This observation showed that the use of a dummy variable to assess the effect of stove improvement led to a clear violation of the proportional hazards assumption, that is, the goodness of model fit was clearly not optimal with the inclusion of such a dummy variable. Therefore, in final models for men and women, the effect of stove improvement was assessed not with a dummy variable but rather with a time-dependent variable whose value switched from zero to one at the age when the subject changed to a stove with a chimney. This procedure gave an overall risk ratio (relative risk or RR) for lung cancer associated with stove improvement, compared with an RR of 1 without improvement. This procedure also improved the fit of the analytical models.

Cox models for men and women also included indicator (dummy) variables for the following: 1) positive lung cancer history in the subject's spouse (to control partially for potential environmentally related nonindependence among observations); 2) positive lung cancer history in the subject's parents, siblings, or children (a known lung cancer risk factor providing partial control for potential biologically related nonindependence); 3) having five people or more in the household at time of exit from follow-up compared with fewer than five people (to adjust partially for cooking load and for possible family-related nonindependence among observations); 4) reporting diagnosed chronic bronchitis or emphysema (often observed to be a lung cancer risk factor); 5) reporting diagnosed tuberculosis [often observed to be a lung cancer risk factor *(14,15)*]; 6) spending 7 daily waking hours or more indoors through age 20 years (to adjust for elevated exposure to indoor coal smoke early in life); 7) using an annual average of more than 3 tons of coal in the household (to adjust for elevated lifetime exposure to indoor coal smoke); 8) being born outside the study area (to adjust for avoidance of early indoor exposure to smoke from coal mined in the study area, although not to smoke from coal mined elsewhere); 9) having any education (to adjust for socioeconomic status); 10) having three or more rooms in the home at exit from follow-up, compared with fewer than three rooms (to adjust for socioeconomic status and for potential effects of coal smoke dilution in the home); and 11) six dummy variables for being born in the following years 1922–1926, 1927–1931, 1932–1936, 1937–1941, 1942–1946, and 1947–1951, compared with 1917–1921 (to adjust for potential birth cohort effects).

The men's model included the following additional covariates: three time-dependent variables for different durations of smoking up to the age at each successive risk set comparison (20–29 years, 30–39 years, or 40 years or more of smoking), relative to a reference category of men who had not smoked or had smoked for less than 20 years; a time-dependent covariate for the effect of cooking food for 20 years or more, relative to a reference category of men who had not cooked food or had cooked for less than 20 years; and a dummy variable for ever being a coal miner, observed to be a lung cancer risk factor in preliminary analyses.

The women's model included three time-dependent covariates for different durations of cooking food up to the age at each successive risk set comparison (20–29 years, 30–39 years, or 40 years or more of cooking), relative to a reference category of women who had not cooked food or had cooked for less than 20 years. Only 87 women (0.9%) had ever smoked, and so the women's model was not adjusted for smoking.

During the time of this study, rural Chinese communes were typically divided geographically into eight to 15 administrative subunits called "large teams." Large teams were further divided into about 10–15 small teams. Residential stability is high in Xuanwei, so that extended families were generally concentrated in single teams. To control partially for potential nonindependence among observations arising from presence of multiple family members in the dataset, the main Cox models were stratified on large team (total 25 strata). Supplemental models were stratified on small team (298 strata for men and 307 strata for women). We elected to stratify the main models on large team because many small teams contained very few subjects.

Alternative sex-specific Cox models were stratified on 5-year birth cohorts. These models included 24 dummy variables for separate large teams as covariates. Other covariates were identical to those in the main models.

Additional sex-specific Cox models were constructed to characterize the time course of the incidence rate of lung cancer after stove improvement. Subjects who changed to a stove with a chimney less than 10 years, 10–19 years, and 20 years or more before leaving follow-up were assigned to separate groups. For each group, a time-dependent variable, whose value switched from zero to one at the age at stove improvement, was defined. This procedure gave a modeled RR for lung cancer after stove improvement in each group, compared with the group with no stove improvement. In these models, all other independent variables and stratification were identical to those in the main models. These models were tested with all subjects included and then with successive exclusion of subjects who left follow-up within 1 year and 2 years after stove improvement. All reported statistical tests were two-sided.

Indoor Air Sampling

In 1995, household indoor air pollution concentrations were measured during unvented and vented smoky coal burning in the study area. Concentrations of particulate matter with a diameter of 10 μm or less (PM_{10}) and of benzo[*a*]pyrene were measured in 15 homes during normal activities. This study was approved by the Institutional Review Board of the Chinese Academy of Preventive Medicine, and written informed consent was obtained from the heads of all 15 households. Two homes had unvented firepits and 13 homes had stoves with chimneys. In these 13 homes, the chimney was open for 2 days and blocked for 3 days. In each home, air sampling was conducted 24 hours/day for 5 consecutive days. Samplers, which drew air at 4 liters/minute, were located about 1.2 meters above the floor and about 1.2 meters laterally from the firepit or stove. Samples were collected on glass fiber filters. Concentrations of PM_{10} were determined gravimetrically. Filters from each home were then pooled into 28 samples (two samples for firepits, 13 for chimneys blocked, and 13 for chimneys open). Benzo[*a*]pyrene concentrations were determined by high-pressure liquid chromatography of each pooled sample. Average pollutant levels during unvented burning (firepit and chimneys blocked) were compared with average levels during vented burning (chimneys open).

Results

Table 1 shows characteristics of the 21 232 subjects included in this analysis. Characteristics with similar sex distributions are shown for men and women combined. After birth, 4048 subjects (19.1%) had no stove improvement and 17 184 (80.9%) changed permanently to stoves with chimneys. There were 1384 lung cancer diagnoses during follow-up, 700 in men (cumulative incidence = 6.3%) and 684 in women (cumulative incidence = 6.8%). Crude cumulative lung cancer incidence was 3.2 times higher in subjects with no stove improvement (14.6%) than in subjects with improvement (4.6%). Age-adjusted lung cancer incidences were 383 and 554 per 100 000 person-years in subjects with and without stove improvement, respectively. Cell type was determined in 182 patients with a lung cancer diagnosis (13.2%).

The percentage of men (6.1%) reporting lung cancer in parents, siblings, or children was somewhat higher than the corresponding percentage of women (4.5%). One hundred one men (0.9%) and 1311 women (13.0%) were born outside the study area; of these 1412 subjects, only 11 were born outside Xuanwei. Most subjects born outside the study area were women who moved into their husbands' households upon marriage when they were 20–25 years old. The average level of education was grade school. Men were better educated than women, and subjects with stove improvement were somewhat better educated than those without it. Smoking and cooking histories did not differ substantially between subjects with and without stove improvement.

Fig. 1 shows product-limit curves. In both men and women, age-specific probabilities of remaining lung cancer-free were distinctly higher in subjects who changed to stoves with chimneys than in those who did not change. In both sexes, differences between curves for subjects with and without stove improve-

Table 1. Characteristics of cohort members included in analysis, by stove use history in Xuanwei, 1976–1992*

	No stove improvement	Changed to stove with chimney	Total
Men and women combined			
Total included in data analysis, No. (%)	4048 (100.0)	17 184 (100.0)	21 232 (100.0)
Age as of January 1, 1976, y (CI)	42.3 (42.0 to 42.7)	38.2 (38.0 to 38.3)	39.0 (38.8 to 39.1)
Age at exit from follow-up, y (CI)	54.0 (53.7 to 54.3)	53.7 (53.6 to 53.8)	53.7 (53.6 to 53.9)
Lung cancer diagnoses, 1976–1992, No. (%)	592 (14.6)	792 (4.6)	1384 (6.5)
Age-adjusted lung cancer incidence per 100 000 person-years, No. (%)	554 N/A	383 N/A	441 N/A
Died without lung cancer during follow-up, No. (%)	1414 (35.0)	2128 (12.4)	3542 (16.7)
Alive and lung cancer-free in 1992, No. (%)	2039 (50.4)	14 267 (83.0)	16 306 (76.8)
Age at lung cancer diagnosis, y (CI)	53.1 (52.4 to 53.8)	54.3 (53.7 to 54.9)	53.8 (53.3 to 54.2)
Lung cancer cell type, No. (%)†			
Squamous cell	53 (9.0)	62 (7.8)	115 (8.3)
Adenocarcinoma	24 (4.1)	25 (3.2)	49 (3.5)
Small cell or mixed cell	9 (1.5)	9 (1.1)	18 (1.3)
Unknown	506 (85.5)	696 (87.9)	1202 (86.8)
Age at stove improvement, y (CI)	N/A N/A	41.9 (41.7 to 42.1)	N/A N/A
Time from stove improvement until exit from follow-up, y (CI)	N/A N/A	11.8 (11.7 to 11.9)	N/A N/A
Lung cancer in spouse, No. (%)	187 (4.6)	706 (4.1)	893 (4.2)
People in household, No. (CI)	5.4 (5.3 to 5.4)	5.1 (5.1 to 5.1)	5.2 (5.1 to 5.2)
Born in 1937–1951, No. (%)	1662 (41.1)	9777 (56.9)	11 439 (53.9)
Chronic bronchitis or emphysema history, No. (%)	996 (24.6)	1323 (7.7)	2319 (10.9)
Tuberculosis history, No. (%)	70 (1.7)	89 (0.5)	159 (0.7)
People with ⩾7 waking h indoors per day through age 20 y, No. (%)	2537 (62.7)	11 309 (65.8)	13 846 (65.2)
People who used average >3 tons of fuel per y, No. (%)	1623 (40.1)	11 697 (68.1)	13 320 (62.7)
Rooms in home, No. (CI)	1.8 (1.8 to 1.9)	1.7 (1.7 to 1.7)	1.7 (1.7 to 1.8)
Men only			
Total No. (%)‡	2160 (100.0)	9008 (100.0)	11 168 (100.0)
Lung cancer in parents, siblings, or children, No. (%)	128 (5.9)	556 (6.2)	684 (6.1)
No. born outside the study area (%)	19 (0.9)	82 (0.9)	101 (0.9)
No. with any education (%)	852 (39.4)	4354 (48.3)	5206 (46.6)
No. ever a coal miner (%)	156 (7.2)	880 (9.8)	1036 (9.3)
No. ever smoked tobacco (%)	1909 (88.4)	8357 (92.8)	10 266 (91.9)
Years of smoking in smokers (CI)	32.7 (32.2 to 33.1)	32.0 (31.8 to 32.3)	32.2 (32.0 to 32.4)
Age at starting to smoke, y (CI)	21.2 (21.0 to 21.3)	21.6 (21.5 to 21.7)	21.5 (21.5 to 21.6)
No. of cigarettes plus tobacco equivalents smoked per day (CI)	17.8 (17.4 to 18.3)	19.3 (19.1 to 19.5)	19.0 (18.9 to 19.2)
No. ever quitting smoking (%)	32 (1.7)	128 (1.5)	160 (1.4)
No. ever cooked food (%)	324 (15.0)	668 (7.4)	992 (8.9)
Years of cooking in men who cooked (CI)	28.6 (27.0 to 30.1)	27.5 (26.4 to 28.6)	27.8 (27.0 to 28.7)
Age at starting to cook, y (CI)	25.5 (24.0 to 27.1)	28.0 (26.8 to 29.2)	27.2 (26.2 to 28.2)
Women only			
Total No. (%)‡	1888 (100.0)	8176 (100.0)	10 064 (100.0)
Lung cancer in parents, siblings, or children, No. (%)	80 (4.2)	368 (4.5)	448 (4.5)
No. born outside the study area (%)	246 (13.0)	1065 (13.0)	1311 (13.0)
No. with any education (%)	364 (19.3)	2008 (24.6)	2372 (23.6)
No. ever smoked tobacco (%)	25 (1.3)	62 (0.8)	87 (0.9)
No. ever cooked food (%)	1826 (96.7)	8001 (97.9)	9827 (97.6)
Years of cooking in women who cooked (CI)	37.1 (36.6 to 37.6)	34.8 (34.5 to 35.0)	35.2 (35.0 to 35.4)
Age at starting to cook, y (CI)	16.6 (16.4 to 16.9)	18.1 (18.0 to 18.2)	17.8 (17.8 to 17.9)

*Data are expressed as the number or the mean value (% or 95% confidence interval [CI]). Numbers (counts) are given as integers. Means are given to one decimal place. N/A = not applicable.

†Percentages for cell type categories are of all lung cancer diagnoses in the designated stove use group.

‡Percentages and 95% confidence intervals are given by sex.

ment were highly statistically significant (P<.001), as indicated by Wilcoxon, log-rank, and likelihood-ratio χ^2 statistics.

The main Cox models are summarized in Table 2. In both sexes, changing to stoves with chimneys was highly statistically significantly associated with a reduced incidence rate of lung cancer (RR = 0.59, 95% confidence interval [CI] = 0.49 to 0.71 for men; RR = 0.54, 95% CI = 0.44 to 0.65 for women; P<.001 for both). Positive histories of lung cancer in the spouse and in biologic family members were both strongly associated with an increased incidence of lung cancer, as was personal history of chronic bronchitis or emphysema. A personal history of tuberculosis was modestly associated with an increased incidence rate. The presence of five people or more in the household was strongly associated with an increased incidence rate.

For both sexes, spending substantial time indoors early in life was statistically significantly associated with increased lung cancer incidence rate, but using an annual average of more than 3 tons of coal was not. Being born outside the study area was associated with markedly reduced incidence. The presence of three rooms or more in the home was statistically significantly associated with a reduced incidence in men (P = .016) but not in women (P = .500). Birth cohort effects were not substantial.

The duration of cooking food was positively and statistically significantly associated with the incidence rate of lung cancer in both sexes. In women, cooking duration exhibited a clear dose–response relationship with incidence. Too few men cooked food to allow reliable testing for a dose–response relationship. Even so, the modeled RR for lung cancer incidence was statistically significantly elevated in men who had cooked food for 20 years or more (P = .023).

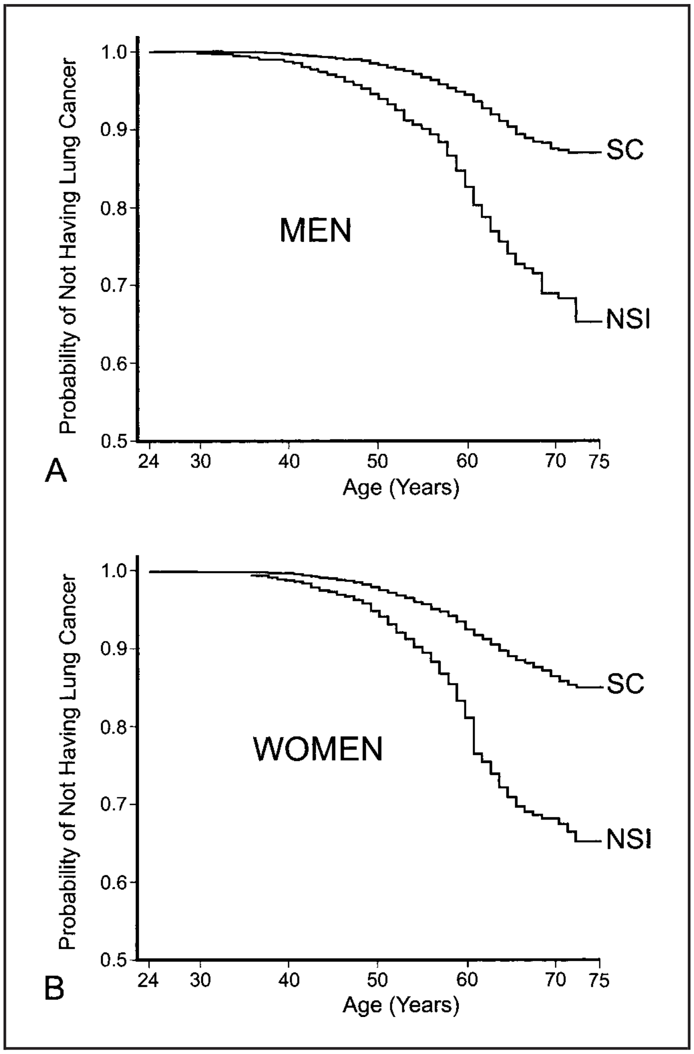

Fig. 1. Product-limit survival plots showing the probability of not having lung cancer, by age in years, in men (**panel A**) and women (**panel B**) who had no stove improvement (NSI) and who changed to stoves with chimneys (SC), in Xuanwei, 1976–1992. Note that the *y* axis (probability) starts at 0.5, not 0. For men at age 45.5 years without stove improvement, the 95% confidence interval (CI) was 0.954 to 0.971 for 1630 subjects at risk (SAR). For men at age 45.5 years with stove improvement, the 95% CI was 0.990 to 0.994 for 6688 SAR. For men at age 65.5 years without stove improvement, the 95% CI was 0.688 to 0.752 for 269 SAR. For men at age 65.5 years with stove improvement, the 95% CI was 0.881 to 0.905 for 1363 SAR. For women at age 45.5 years without stove improvement, the 95% CI was 0.961 to 0.977 for 1424 SAR. For women at age 45.5 years with stove improvement, the 95% CI was 0.988 to 0.992 for 5852 SAR. For women at age 65.5 years without stove improvement, the 95% CI was 0.654 to 0.724 for 224 SAR. For women at age 65.5 years with stove improvement, the 95% CI was 0.871 to 0.897 for 1013 SAR.

In men, smoking exhibited a limited time-dependent dose–response relationship with the incidence of lung cancer that was considerably weaker than that observed in women for cooking. The association of smoking duration with the incidence of lung cancer was statistically significant only with 40 years or more of smoking (RR = 1.49, 95% CI = 1.02 to 2.18, P = .041).

In the supplemental Cox models, which were stratified on small team, RRs of stove improvement for the incidence rate of lung cancer were slightly greater than in the main models, but still highly statistically significant (RR = 0.63 and 0.59 for men and women, respectively; P<.001 for both). In the alternate Cox models that were stratified on birth cohort and that included dummy variables for large teams, RRs of stove improvement were slightly smaller than those in the main models (RR = 0.57 and 0.50 for men and women, respectively; P<.001 for both).

Table 3 summarizes the data from Cox models constructed to characterize the time course of the incidence rate for lung cancer after stove improvement. Modeled RRs of stove improvement consistently decreased as the time interval from improvement until exit from follow-up increased. These RRs were always highly statistically significantly less than 1 in subjects for whom this interval was 10 years or longer (P<.001 in all cases). When this interval was 20 years or longer, the modeled RR in men (RR = 0.07, 95% CI = 0.03 to 0.17) was less than half that in women (RR = 0.17, 95% CI = 0.10 to 0.31). In men and women for whom this interval was less than 10 years, modeled RRs were statistically significantly greater than 1. When subjects who left follow-up 2 years or less after stove improvement were excluded from analysis, the modeled RR for the first 10 years after stove improvement did not differ statistically significantly from 1 in either men or women.

Table 4 shows average levels of indoor air pollution during unvented and vented smoky coal burning in study area homes. Levels of air pollution during vented burning were much lower than during unvented burning. Average concentrations of PM_{10} and benzo[*a*]pyrene during vented burning (0.71 mg/m^3 and 0.25 μg/m^3) were, respectively, only 34.1% and 15.1% of corresponding concentrations during unvented burning (2.08 mg/m^3 and 1.66 μg/m^3).

Discussion

In all analyses, changing from unvented firepits to stoves with chimneys was strongly associated with a reduction in the incidence rate of lung cancer in users of Xuanwei smoky coal. In the following discussion, we address potential sources of artefact in these results.

In rural China, there is much cultural resistance to invasive medical procedures including biopsy, surgery, and autopsy. Only 13.2% of lung cancer diagnoses were cytologically confirmed. Although more complete confirmation would have been desirable, we believe that the degree of diagnostic misclassification was small and that any such misclassification would have had little effect on the analytical results. Among the 1384 patients diagnosed with lung cancer, 1353 (97.8%) died before follow-up ended, and 1277 (92.3%) died within 3 years of diagnosis. Mean times from diagnosis to death also were very similar in patients with known and unknown cell types of lung cancer (1.15 ± 0.21 years and 1.07 ± 0.08 years, respectively). In Cox models that excluded patients with lung cancer who did not die within 1 year of diagnosis, RRs of stove improvement were actually smaller than in the main models (RR = 0.47, P<.001 in men; RR = 0.43, P<.001 in women). Furthermore, in patients diagnosed with lung cancer, the mean time from diagnosis to death or end of follow-up (1.12 ± 0.08 years) was considerably shorter than the corresponding time in subjects who reported chronic bronchitis or emphysema (8.80 ± 0.29 years). Thus, it is unlikely that chronic obstructive lung disease was misclassified as lung cancer to a substantial degree.

The analytical dataset contained members of the same families, leading to some degree of nonindependence among individual observations. Individual families were not specifically identified, so such nonindependence could not be addressed di-

Table 2. Stratified Cox model risk ratios (relative risks or RRs) for lung cancer incidence rate, 95% confidence intervals (CIs) around risk ratios, and *P* values, by sex, in Xuanwei, 1976–1992

	Men*		Women*	
	RR (95% CI)†	*P* value	RR (95% CI)†	*P* value
Independent variables				
No stove improvement	1† N/A		1† N/A	
Changed to stove with chimney	0.59 (0.49 to 0.71)	<.001	0.54 (0.44 to 0.65)	<.001
No lung cancer in spouse	1† N/A		1† N/A	
Lung cancer in spouse	1.51 (1.17 to 1.96)	.002	2.18 (1.72 to 2.77)	<.001
No lung cancer in parents, siblings, or children	1† N/A		1† N/A	
Lung cancer in parents, siblings, or children	2.29 (1.80 to 2.90)	<.001	1.79 (1.33 to 2.40)	<.001
<5 people in household	1† N/A		1† N/A	
⩾5 people in household	2.26 (1.88 to 2.70)	<.001	2.54 (2.10 to 3.06)	<.001
No chronic bronchitis or emphysema	1† N/A		1† N/A	
Chronic bronchitis or emphysema	1.67 (1.37 to 2.04)	<.001	1.37 (1.11 to 1.69)	.003
No tuberculosis	1† N/A		1† N/A	
Tuberculosis	1.69 (0.86 to 3.30)	.127	1.58 (0.86 to 2.91)	.142
<7 waking hours/day indoors through age 20 y	1† N/A		1† N/A	
⩾7 waking hours/day indoors through age 20 y	1.34 (1.11 to 1.61)	.002	1.67 (1.36 to 2.04)	<.001
Used annual average ⩽3 tons of fuel	1† N/A		1† N/A	
Used annual average >3 tons of fuel	0.97 (0.81 to 1.17)	.756	1.13 (0.93 to 1.36)	.221
Born in study area	1† N/A		1† N/A	
Born outside study area	0.34 (0.11 to 1.05)	.061	0.49 (0.35 to 0.67)	<.001
Had no education	1† N/A		1† N/A	
Had education	1.00 (0.84 to 1.19)	.992	1.52 (1.20 to 1.92)	<.001
<3 rooms in home	1† N/A		1† N/A	
⩾3 rooms in home	0.72 (0.56 to 0.94)	.016	0.92 (0.72 to 1.18)	.500
Born 1917–1921	1† N/A		1† N/A	
Born 1922–1926	1.28 (0.99 to 1.65)	.060	1.16 (0.88 to 1.52)	.303
Born 1927–1931	1.25 (0.94 to 1.67)	.130	1.02 (0.76 to 1.38)	.890
Born 1932–1936	1.00 (0.69 to 1.44)	.978	1.29 (0.90 to 1.86)	.173
Born 1937–1941	0.91 (0.59 to 1.41)	.686	1.09 (0.71 to 1.69)	.683
Born 1942–1946	0.88 (0.52 to 1.51)	.651	0.99 (0.58 to 1.69)	.973
Born 1947–1951	0.83 (0.45 to 1.56)	.567	0.92 (0.49 to 1.74)	.804
Men only				
Smoked <20 y‡§	1† N/A			
Smoked 20–29 y	0.87 (0.65 to 1.17)	.352		
Smoked 30–39 y	1.16 (0.85 to 1.59)	.354		
Smoked ⩾40 y	1.49 (1.02 to 2.18)	.041		
Cooked food <20 y§‖	1† N/A			
Cooked ⩾20 years	1.42 (1.05 to 1.93)	.023		
Never a coal miner	1† N/A			
Ever a coal miner	1.40 (1.07 to 1.83)	.014		
Women only				
Cooked food <20 y‖			1† N/A	
Cooked 20–29 y			1.38 (0.87 to 2.20)	.168
Cooked 30–39 y			1.79 (1.09 to 2.93)	.021
Cooked ⩾40 y			3.08 (1.80 to 5.26)	<.001

*In men, there were 165 136.5 person-years during follow-up (65 981.2 before stove improvement and 99 155.4 after stove improvement). In women, there were 148 442.3 person-years during follow-up (58 381.5 before stove improvement and 90 060.8 after stove improvement). The likelihood-ratio χ^2 statistic was 239.6 for the men's model (17 degrees of freedom [*df*], *P*<.001) and 300.8 for the women's model (15 *df, P*<.001). There were 700 diagnosed lung cancer cases in men (6.3%) and 648 cases in women (6.4%). All statistical tests were two-sided. N/A = not applicable.

†Reference group.

‡Includes nonsmokers.

§Time-independent duration of men's smoking and cooking and women's cooking from age at starting to smoke or cook to age at each successive risk set comparison.

‖Includes subjects who did not cook food.

rectly in the data analysis. Even so, we took considerable care to address this issue, albeit indirectly, in our modeling strategy. We also believe it extremely unlikely that an artefact arising from family-related clustering could have explained the observed beneficial effect of stove improvement. We used sex-specific models, and this procedure would have removed any such artefact related to the presence of spouses and biologic family members of different sexes. Our main models also were stratified on large teams, in which extended families would have clustered. The models also included several family-related covariates, such as number of rooms and people in the home and the history of lung cancer in family members.

Table 3. Stratified Cox model risk ratios (relative risks or RRs) for lung cancer incidence rate, by time from stove improvement until leaving follow-up, when excluding subjects with successively longer times after stove improvement, Xuanwei, 1976–1992

	Men		Women	
Subjects excluded	RR* (95% CI)*	Person-years	RR* (95% CI)*	Person-years
No exclusion (all subjects)				
No stove improvement	1† N/A	65 981‡	1† N/A	58 382‡
0–9 y after improvement	1.79 (1.48 to 2.18)	22 323§	1.41 (1.15 to 1.73)	20 529§
10–19 y after improvement	0.25 (0.19 to 0.31)	67 880§	0.24 (0.19 to 0.31)	61 097§
⩾20 y after improvement	0.07 (0.03 to 0.17)	8953§	0.17 (0.10 to 0.31)	8435§
Left follow-up ⩽1 y after stove improvement				
No stove improvement	1† N/A	65 366‡	1† N/A	57 896‡
1–9 y after improvement	1.38 (1.13 to 1.70)	22 264§	1.14 (0.92 to 1.41)	20 486§
10–19 y after improvement	0.23 (0.18 to 0.29)	67 880§	0.22 (0.17 to 0.28)	61 097§
⩾20 y after improvement	0.07 (0.03 to 0.16)	8953§	0.16 (0.09 to 0.28)	8435§
Left follow-up ⩽2 y after stove improvement				
No stove improvement	1† N/A	64 596‡	1† N/A	57 054‡
2–9 y after improvement	1.22 (0.99 to 1.50)	22 116§	0.96 (0.77 to 1.19)	20 332§
10–19 y after improvement	0.22 (0.17 to 0.27)	67 880§	0.21 (0.16 to 0.27)	61 097§
⩾20 y after improvement	0.06 (0.03 to 0.15)	8953§	0.15 (0.09 to 0.28)	8435§

*RR = risk ratio; 95% CI = 95% confidence interval; N/A = not applicable.

†Reference group.

‡Includes person-years in subjects with no stove improvement and in those with improvement but before it occurred.

§Person-years after stove improvement.

Table 4. Indoor airborne concentrations of particulate matter of diameter ⩽10 μm (PM_{10}) and benzo[*a*]pyrene (BaP) in study area homes, by mode of coal burning, Xuanwei, 1995*

Mode of coal burning	No. of homes	PM_{10} (95% CI),* mg/m^3	BaP (95% CI),* μg/m^3
Unvented	15†	2.08 (1.39 to 2.76)	1.66 (1.16 to 2.16)
Vented	13‡	0.71 (0.50 to 0.92)	0.25 (0.03 to 0.47)

*CI = confidence interval.

†Includes two homes with unvented firepits and 13 homes with chimneys blocked.

‡Includes 13 homes with chimneys open.

Furthermore, in the main, supplemental, and alternate Cox models, the beneficial effect of stove improvement was consistently highly statistically significant, with $P<.001$. This effect would have remained statistically significant even if there were a large unadjusted design effect caused by clustering in the data. For example, in the men's main model, the parameter estimate, standard error of the estimate, and χ^2 statistic for stove improvement were –0.5242, 0.0929, and 31.84, respectively. Even with a very large unadjusted design effect of 5, for example, the χ^2 statistic for stove improvement would have been 6.36 ($P = .012$), assuming a constant parameter estimate. For women, a design effect of 5 would have implied a χ^2 statistic of 8.14 ($P = .004$) for stove improvement. Thus, even if our analyses were subject to a very large unadjusted design effect (which we seriously doubt), the beneficial effect of stove improvement would still have been statistically significant.

Data from persons born outside the study area should have been less subject to potential effects of family-related clustering than data from those born inside the study area. In an additional Cox model restricted to the 1311 women born outside the study area, the RR of stove improvement was actually smaller than in the main women's model (RR = 0.37, 95% CI = 0.17 to 0.78; $P = .010$). This finding increases confidence that the associations of stove improvement with reduced lung cancer incidence rate, as observed in all subjects, are valid.

In the main models, RRs of covariates for chronic nonmalignant lung disease and for a family history of lung cancer were highly statistically significant. Chronic lung disease may sometimes be an intermediate event between coal smoke exposure and lung cancer. Also, the large modeled RRs of family history of lung cancer could be attributed partly to common within-family exposure to indoor coal smoke. Thus, the models may be overspecified somewhat, and modeled RRs and 95% CIs are therefore subject to some uncertainty. To assess potential consequences of this uncertainty, the models were re-examined with all possible combinations of these covariates, including exclusion of all (a total of seven additional models for each sex). Modeled RRs of stove improvement were very similar to those in the main models (0.56 to 0.59 in men; 0.52 to 0.54 in women; $P<.001$ for both). Thus, any potential overspecification of the main models would have had no substantial effect on modeled RRs or confidence intervals of stove improvement for the incidence rate of lung cancer.

Duration of cooking food was strongly and positively associated with lung cancer incidence. This association was observed in men and women, although far fewer men than women cooked. Compared with women, men who cooked started cooking later in life and cooked for a shorter time (Table 1). Thus, close proximity to an unvented stove and, consequently, elevated exposure to pollutants in coal smoke appear to increase the risk of lung cancer for residents of Xuanwei, even when such elevated exposure begins in adulthood.

Duration of smoking was positively associated with lung cancer incidence in men, although the effect of cooking in women was stronger than the effect of smoking in men. Indeed, the smoking effect in men was considerably weaker than that observed in most other lung cancer studies *(16–23)*. The high exposure to indoor airborne carcinogens from coal smoke conceivably could overwhelm the effects of smoking in this cohort.

Lung cancer incidence was markedly reduced in subjects born outside the study area. Early in life, these subjects were not exposed to smoke from smoky coal mined in the study area, even though they were exposed to smoky coal smoke in communes outside the study area. Thus, the carcinogenicity of smoky coal smoke may be higher in the study area than elsewhere in Xuanwei and perhaps higher than elsewhere in China.

The presence of five or more people in the household was strongly associated with an increased incidence of lung cancer in both men and women (Table 2). As mentioned above, this covariate provided a test for a possible effect of an increased cooking load in larger households on lung cancer incidence. The observed association suggests that such an increased cooking load and, consequently, an increased indoor exposure to coal smoke are indeed important lung cancer risk factors in Xuanwei. Two additional observations support this interpretation. First, average annual coal consumption in households with at least five people (mean = 3.50 tons, 95% CI = 3.48 to 3.51 tons) was larger than that in smaller households (mean = 3.27 tons, 95% CI = 3.25 to 3.30 tons). Second, in the Cox model for each sex, omitting the covariate for five or more people in the household led to an increase in the modeled RR of using an annual average of more than 3 tons of coal. In women, this RR increased from 1.13 (Table 2) to 1.30 and became statistically significant ($P = .006$).

The presence of three or more rooms in the home was associated with a modeled reduction in the incidence of lung cancer (Table 2). This reduction was statistically significant in men ($P = .016$) but not in women ($P = .500$). The degree of indoor coal smoke dilution would be expected to increase, and consequent coal smoke exposure would be expected to decrease, with an increasing number of rooms in the home. If such exposure were etiologically related to lung cancer, an inverse association of the RR for lung cancer with the number of rooms (such as we observed) would be expected. In addition, as mentioned above, far fewer men than women cooked food. Thus, men probably had more opportunity than women to spend time away from the stove, where airborne coal smoke was presumably more diluted than it was at the stove. In view of this observation, it is not at all surprising that the modeled beneficial effect of a large number of rooms was stronger in men than in women. Thus, these observations tend to confirm the importance of exposure to indoor smoky coal smoke in the etiology of lung cancer in Xuanwei.

Surrogate respondents were interviewed for 1368 (98.8%) of the 1384 lung cancer patients, for all of the 3542 subjects who died without lung cancer, and for 5314 (32.6%) of the 16 306 subjects who remained alive and free of lung cancer. Among the 10 224 surrogates, 5302 (51.8%) were subjects' children, siblings, or parents; 3250 (31.8%) were spouses; and 1672 (16.4%) were close friends, usually next-door neighbors. Such surrogates would have been highly familiar with subjects and their activities. Nevertheless, surrogate-related recall bias could conceivably have affected the analytical results. To assess this possibility, Cox models were constructed with only surrogates included. Modeled RRs of stove improvement were 0.54 ($P<.001$) for men and 0.50 ($P<.001$) for women. Thus, for surrogates, as for all subjects, stove improvement was clearly associated with a reduced incidence of lung cancer, and it is very unlikely that the present results could be explained by recall bias.

Previous studies have shown that the risk of lung cancer decreases after cessation of smoking *(16,21,23–26)*. Reported time intervals from cessation to a clear risk reduction have ranged from less than 5 years to 10 years or longer. Our analyses suggested that the time from stove improvement to unequivocal risk reduction was about 10 years. The analyses also suggested that incidence shortly after stove improvement was higher than that without improvement. This observation does not imply that stove improvement conferred short-term increase in lung cancer risk, but rather it is reminiscent of the "quitting-ill effect" observed in smoking-cessation studies *(16,21,24–26)*. The appearance of early lung cancer symptoms spurs some smokers to quit and could similarly have spurred some Xuanwei residents to change stoves before diagnosis. This hypothesis is consistent with the anecdotal evidence, mentioned above—that some subjects changed stoves only after the onset of respiratory symptoms.

We confined our examination of the time course of lung cancer risk reduction to rather wide time intervals. We believe that the data would not support a reliable assessment of shorter intervals. Specifically, among subjects with stove improvement, the questionnaire interview was conducted an average of 12.8 years after the reported age at stove improvement (range = 0.03–55.6 years). Such a long recall interval lends uncertainty to the reported age at stove improvement, particularly because a substantial proportion of respondents were surrogates. Even relatively small differences in the reported age at improvement would generate substantial proportional differences in the number of person-years within time intervals shorter than about 10 years. Such differences also would create instability in the proportions of person-years with and without improvement, leading in turn to instability of modeled RRs within these shorter intervals.

At the same time, we emphasize that the modeled overall effect of stove improvement would have been strongly beneficial, even if there had been a large systematic error in the reported age at stove improvement. For example, across a systematic error range of plus or minus 5 years, the modeled RR of improvement would have ranged from 0.30 to 0.73 ($P \leq .001$) in men and from 0.25 to 0.75 ($P \leq .003$) in women. Even across such a wide range, lung cancer risk still would have been unequivocally reduced 10 years and longer after stove improvement.

The 1995 indoor air pollution study realistically simulated pollutant concentrations in the homes of cohort members with and without stove improvement. These concentrations were far higher during unvented burning than during burning with open chimneys. This study indicates that use of chimneys markedly reduces indoor air pollution concentrations in study area homes.

In summary, our results strongly support the hypothesis that stove improvement reduces indoor air pollution exposure and thereby reduces the risk of lung cancer in users of Xuanwei smoky coal. Indoor air pollution is a serious health hazard in developing countries *(1,2,4,27,28)*. Coal smoke exposure is associated with lung cancer *(29–33)* and nonmalignant respiratory disorders *(34,35)*. Exposure to smoke from biomass fuel is associated mainly with nonmalignant disorders, including acute lower respiratory illness in children *(4,5)*, chronic respiratory illness and symptoms in adults (especially women) *(6,36,37)*, pulmonary hypertension and consequent right-sided heart disease (cor pulmonale) *(38)*, lung function changes *(36,39)*, and possibly eye disorders *(40)* and adverse pregnancy outcomes *(41)*. The population at risk for illness mediated by indoor air pollution numbers hundreds of millions in China alone *(42)* and possibly billions worldwide *(43)*. The present study and previous studies indicate that stove improvements can substantially reduce the levels of indoor air pollution *(7,8)*, if tailored to local conditions *(44,45)*. Thus, available evidence strongly suggests that household stove improvements would benefit public health on a global scale *(29,46,47)*.

To the best of our knowledge, however, previous studies have not documented health benefits of stove improvement in households using coal or biomass fuel. We hope that the documentation presented in this article will stimulate further development and implementation of appropriate stove improvements and of other strategies to reduce indoor air pollution in developing countries. That many millions of people cannot yet change household fuels renders these efforts urgent.

References

(1) Smith KR. Fuel combustion, air pollution exposure, and health: the situation in developing countries. Annu Rev Energy Environ 1993;18:529–66.

(2) Chen BH, Hong CJ, Pandey MR, Smith KR. Indoor air pollution in developing countries. World Health Stat Q 1990;43:127–38.

(3) Mumford JL, He XZ, Chapman RS, Cao SR, Harris DB, Li XM, et al. Lung cancer and indoor air pollution in Xuan Wei, China. Science 1987;235: 217–20.

(4) Smith KR, Samet JM, Romieu I, Bruce N. Indoor air pollution in developing countries and acute lower respiratory infections in children. Thorax 2000;55:518–32.

(5) Pandey MR, Neupane RP, Gautam A, Shrestha IB. Domestic smoke pollution and acute respiratory infections in a rural community of the hill region of Nepal. Environ Int 1989;15:337–40.

(6) 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 Pt 1):701–6.

(7) Pandey MR, Neupane RP, Gautam A, Shrestha IB. The effectiveness of smokeless stoves in reducing indoor air pollution in a rural hill region of Nepal. Mountain Res Dev 1990;10:313–20.

(8) Naeher LP, Smith KR, Leaderer BP, Grajeda R, Mage D, Boleij JS. Particulates and CO in highland Guatemala: indoor and outdoor levels from traditional and improved wood stoves and gas stoves in three test households. In: Indoor Air '96. The 7th International Conference on Indoor Air Quality and Climate; 1996 July 21–26; Nagoya (Japan) pp. 405–10.

(9) Li J, Liu B, Li GY, and the Editorial Committee for the Atlas of Cancer Mortality in the People's Republic of China. Atlas of cancer mortality in the People's Republic of China. Shanghai (China): China Map Press; 1979.

(10) Mumford JL, Chapman RS, Harris DB, He XZ, Cao SR, Xian YL, Li XM. Indoor air exposure to coal and wood combustion emissions associated with a high lung cancer rate in Xuan Wei, China. Environ Int 1989;15: 315–20.

(11) He XZ, Chen W, Liu ZY, Chapman RS. An epidemiological study of lung cancer in Xuan Wei County, China: current progress. Case–control study on lung cancer and cooking fuel. Environ Health Perspect 1991;94:9–13.

(12) Liang CK, Quan NY, Cao SR, He XZ, Ma F. Natural inhalation exposure to coal smoke and wood smoke induces lung cancer in mice and rats. Biomed Environ Sci 1988;1:42–50.

(13) Mumford JL, Helmes CT, Lee XM, Seidenberg J, Nesnow S. Mouse skin tumorigenicity studies of indoor coal and wood combustion emissions from homes of residents in Xuan Wei, China with high lung cancer mortality. Carcinogenesis 1990;11:397–403.

(14) Zheng W, Blot WJ, Liao ML, Wang ZX, Levin LI, Zhao JJ, et al. Lung cancer and prior tuberculosis infection in Shanghai. Br J Cancer 1987;56: 501–4.

(15) Ernster VL. Female lung cancer. Annu Rev Public Health 1996;17:97–114.

(16) Doll R, Peto R. Mortality in relation to smoking: 20 years' observations on male British doctors. Br Med J 1976;2:1525–36.

(17) Gao YT, Blot WJ, Zheng W, Fraumeni JF, Hsu CW. Lung cancer and smoking in Shanghai. Int J Epidemiol 1988;17:277–80.

(18) Liu Z. Smoking and lung cancer in China: combined analysis of eight case–control studies. Int J Epidemiol 1992;21:197–201.

(19) Lubin JH, Li JY, Xuan XZ, Cai SK, Luo QS, Yang LF, et al. Risk of lung cancer among cigarette and pipe smokers in southern China. Int J Cancer 1992;51:390–5.

(20) Samet JM. The epidemiology of lung cancer. Chest 1993;103(1 Suppl): 20S–29S.

(21) Halpern MT, Gillespie BW, Warner KE. Patterns of absolute risk of lung cancer mortality in former smokers. J Natl Cancer Inst 1993;85:457–64.

(22) Doll R, Peto R, Wheatley K, Gray R, Sutherland I. Mortality in relation to smoking: 40 years' observations on male British doctors. BMJ 1994;309: 901–11.

(23) Peto R, Darby S, Deo H, Silcocks P, Whitley E, Doll R. Smoking, smoking cessation, and lung cancer in the UK since 1950: combination of national statistics with two case–control studies. BMJ 2000;321:323–9.

(24) Lubin JH, Blot WJ, Berrino F, Flamant R, Gillis CR, Kunze M, et al. Modifying risk of developing lung cancer by changing habits of cigarette smoking. Br Med J (Clin Res Ed) 1984;288:1953–6.

(25) Higgins IT, Wynder EL. Reduction in risk of lung cancer among ex-smokers with particular reference to histologic type. Cancer 1988;62: 2397–401.

(26) Kawachi I, Colditz GA, Stampfer MJ, Willett WC, Manson JE, Rosner B, et al. Smoking cessation in relation to total mortality rates in women. A prospective cohort study. Ann Intern Med 1993;119:992–1000.

(27) de Koning HW, Smith KR, Last JM. Biomass fuel combustion and health. Bull World Health Organ 1985;63:11–26.

(28) Florig HK. China's air pollution risks. Environ Sci Technol 1997;31: 274A–9A.

(29) Smith KR, Liu YC. Indoor air pollution in developing countries. In: Samet JM, editor. Epidemiology of lung cancer. Lung biology in health and disease series. Vol 74. New York (NY): Marcel Dekker; 1994. p. 151–84.

(30) Wu-Williams AH, Dai XD, Blot W, Xu ZY, Sun XW, Xiao HP, et al. Lung cancer among women in north-east China. Br J Cancer 1990;62:982–7.

(31) Xu ZY, Blot WJ, Li G, Fraumeni JF Jr, Zhao DZ, Stone BJ, et al. Environmental determinants of lung cancer in Shenyang, China. IARC Sci Publ 1991;(105):460–5.

(32) Tao XG, Hong CJ, Yu SZ, Zhu HG. Risk of male lung cancer attributed to coal combustion indoors in Shanghai. Public Health Rev 1991–92;19(1–4): 127–34.

(33) Luo RX, Wu B, Yi YN, Huang ZW, Lin RT. Indoor burning coal air pollution and lung cancer—a case–control study in Fuzhou, China. Lung Cancer 1996;14 Suppl 1:S113–9.

(34) Hu HS, Liu YC. Evaluation of indoor air pollution and its effect on human health in Beijing's China rural areas. Environ Int 1989;15:321–36.

(35) Tao XG, Hong CJ, Yu SZ, Chen BH, Zhu HG, Yang MD. Priority among air pollution factors for preventing chronic obstructive pulmonary disease in Shanghai. Sci Total Environ 1992;127:57–67.

(36) Norboo T, Yahya M, Bruce NG, Heady JA, Ball KP. Domestic pollution and respiratory illness in a Himalayan village. Int J Epidemiol 1991;20: 749–57.

(37) Ellegard A. Cooking fuel smoke and respiratory symptoms among women in low-income areas in Maputo. Environ Health Perspect 1996;104:980–5.

(38) 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;103:12–20.

(39) Pandey MR, Regmi HN, Neupane RP, Gautam A, Bhandari DP. Domestic smoke pollution and respiratory function in rural Nepal. Tokai J Exp Clin Med 1985;10:471–81.

(40) Mohan M, Sperduto RD, Angra SK, Milton RC, Mathur RL, Underwood BA, et al. India–US case–control study of age-related cataracts. India–US Case–Control Study Group. Arch Ophthalmol 1989;107:670–6.

(41) Mavalankar DV, Gray RH, Trivedi CR. Risk factors for preterm and term low birthweight in Ahmedabad, India. Int J Epidemiol 1992;21:263–72.

(42) Sinton JE, Smith KR, Hu HS, Liu JZ. Indoor air pollution database for China. Geneva (Switzerland): World Health Organization; 1995. WHO/ EHG/95.8.

(43) Health and environment in sustainable development five years after the earth summit. Geneva (Switzerland): World Health Organization; 1997. WHO/EHG/97.8.

(44) Pandey MR, Boleij JS, Smith KR, Wafula EM. Indoor air pollution in developing countries and acute respiratory infection in children. Lancet 1989;1:427–9.

(45) Ramakrishna J, Durgaprasad MB, Smith KR. Cooking in India: the impact of improved stoves on indoor air quality. Environ Int 1989;15:341–52.

(46) Indoor air pollution in developing countries. Lancet 1990;336:1548.

(47) Indoor air pollution and acute respiratory infections in children. Lancet 1992;339:396–8.

Notes

Q. Lan and R. Chapman contributed equally to this report.

Supported by the Chinese Academy of Preventive Medicine, Beijing, China, by the Yunnan Province Antiepidemic Station, Kunming, China, and by contract No. 5D2290NFFX from the U.S. Environmental Protection Agency.

We are especially grateful to the Xuanwei residents who participated in the research. This study would not have been possible without the cooperation of many Chinese administrative and public health officials, physicians, and survey workers. We are grateful to Chaofu Huang of the Xuanwei County Antiepidemic Station, Rongfa Li of the Qujing District Antiepidemic Station, and Rudao Yang and Zhiyu Jin of the Yunnan Province Antiepidemic Station in Kunming. D. Bruce Harris, A. Wayne Fowler, and James W. Montgomery of the U.S. Environmental Protection Agency (Research Triangle Park, NC) designed and built the particulate samplers used in the indoor air sampling study. John Creason of the U.S. Environmental Protection Agency (Research Triangle Park) provided statistical advice during manuscript preparation.

The research described in this paper has been reviewed by the U.S. Environmental Protection Agency. The contents do not necessarily reflect the views or policies of the Agency, nor does mention of trade names or commercial products constitute endorsement or recommendation for use.

Manuscript received September 17, 2001; revised April 2, 2002; accepted April 8, 2002.