Indoor NO₂ air pollution and lung function of professional cooks

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Abstract

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Studies of cooking-generated NO₂ effects are rare in occupational epidemiology. In the present study, we evaluated the lung function of professional cooks exposed to NO₂ in hospital kitchens. We performed spirometry in 37 cooks working in four hospital kitchens and estimated the predicted FVC, FEV_1 and FEF_{25-75} , based on age, sex, race, weight, and height, according to Knudson standards. NO2 measurements were obtained for 4 consecutive days during 4 different periods at 20-day intervals in each kitchen. Measurements were performed inside and outside the kitchens, simultaneously using Palm diffusion tubes. A time/exposure indicator was defined as representative of the cumulative exposure of each cook. No statistically significant effect of NO₂ exposure on FVC was found. Each year of work as a cook corresponded to a decrease in predicted FEV₁ of 2.5% (P = 0.046) for the group as a whole. When smoking status and asthma were included in the analysis the effect of time/exposure decreased about 10% and lost statistical significance. On predicted FEF₂₅₋₇₅, a decrease of 3.5% (P = 0.035) was observed for the same group and the inclusion of controllers for smoking status and asthma did not affect the effects of time/exposure on pulmonary function parameter. After a 10-year period of work as cooks the participants of the study may present decreases in both predicted FEV₁ and FEF₂₅₋₇₅ that can reach 20 and 30%, respectively. The present study showed small but statistically significant adverse effects of gas stove exposure on the lung function of professional cooks.

Key words

- Indoor air pollution
- Occupational diseases

- Lung function
- Nitrogen dioxide
- Professional cooks
- Kitchen

Introduction

Nitrogen dioxide (NO₂) is an air pollutant which is present in both outdoor and indoor environments. NO2 is generated in outdoor environments mainly by vehicles, fossil fuel-burning power plants and industrial boilers, while indoor NO₂ concentrations are affected by outdoor levels, indoor appliances such as gas stoves and kerosene heaters, cigarette smoke, and ventilation of both combustion appliances and indoor environment (1). NO_2 is a deep lung irritant because of its limited solubility and high oxidative potential. Exposures to high concentrations may promote severe acute lung damages and, in some cases, even death (2). Children, the elderly and the ill are the segments of the population more affected by NO2 exposure because of the long periods of time spent indoors (3).

Gas stoves are among the major contributors to indoor NO_2 exposure. Studies conducted in New York (4) and London (5) showed that NO_2 concentrations in the kitchens of homes with gas stoves (49.1 and 72.3 ppb, respectively) were higher than in the kitchens with electric stoves (8.3 and 9.5 ppb, respectively).

The few studies that have examined the relation between the use of gas stoves in homes and respiratory symptoms or lung function in adults have reached different conclusions. Some studies found no association between gas cooking and respiratory symptoms or diminished lung function in adults (6), but other studies have reported associations between gas cooking and an increased prevalence of respiratory symptoms (7) and lung function impairment (8,9).

Smoking has not been a confounder in the association between gas stove exposure and respiratory effects. Helsing et al. (10), using a sample of non-smokers, showed that gas cooking was associated with an increased risk of airway obstruction. Non-smoking women in Singapore who frequently use gas for cooking have more respiratory symptoms and worse lung function than women who use gas infrequently or not at all (11).

Jarvis et al. (12) showed that exposure to NO_2 from gas cooking was associated with decreased respiratory function and increased respiratory symptoms in women but not in men and this difference was attributed to differences in exposure to gas stove appliances between men and women.

In industrial kitchens, cooks are exposed daily to NO_2 emissions from gas stoves and usually for long periods of time and for many years. The effects of this repeated and prolonged exposure on the lung function of cooks have not been fully assessed. Thus, the objective of the present study was to estimate the impact of NO_2 concentrations in industrial kitchens on the respiratory function of professional cooks.

Material and Methods

Pulmonary function measurements

At the time of the study there were 6 hospitals in Araraquara, SP, Brazil. The 4 largest hospitals were selected to participate in the study and all of the 37 people who worked as cooks were included: 8 people from hospital 1, 17 from hospital 2, 8 from hospital 3, and 5 from hospital 4. One person worked simultaneously at 2 hospitals (1 and 2). A detailed time/budget analysis of the population was carried out using a questionnaire for information on age, smoking, time spent in the industrial kitchen (in years), health status (including previous diseases), medication intake, habits out of the kitchen, and socioeconomic status (years of study and income). We also performed lung function tests on all participants, with measurements taken between 12:00 am and 2:00 pm. Spirometry was performed according to the 1987 recommendations of the American Thoracic Society (13). The subjects performed a minimum of 5 and a maximum of 8 attempts to obtain at least three satisfactorily forced expiratory maneuvers. Forced vital capacity (FVC), forced expiratory volume in 1 s (FEV₁), and forced expiratory flow between 25 and 75% of FVC (FEF₂₅₋₇₅) were recorded based on the best FEV1 measurement. To classify tests for reliability, two qualified technicians from the spirometry quality control center of the Physiotherapy Division of Araraquara University reviewed all tests. Only reliable test results were included in the present analysis. Among all cooks selected for the study only one person did not reach reliability standards during his test and the cook was excluded. We used predicted pulmonary function values from Knudson standards (13).

Exposure measurements

The areas of the kitchens were 28.35 m² (hospital 1), 191.4 m² (hospital 2), 75.2 m² (hospital 3), and 25.73 m² (hospital 4). We took NO₂ measurements on 4 consecutive days during 4 different periods separated by 20-day intervals in each kitchen. Measurements were made inside and outside the kitchens, simultaneously using a diffusion tube adapted from that first developed by Palmes et al. (14). The diffusion tube consists of a hollow plastic tube and two closely fitting caps. The plastic tube is 60-mm long and 17 mm in diameter. One of the tube ends is covered with a small plastic mesh coated with 11% triethanolamine.

At the same time, NO_2 concentrations were measured in five places in the city: one in the central bus station in the South region, one downtown, one on the East side of the city, and two in the South-West region. These measurements were made in order to obtain an estimate of the outdoor NO_2 levels in the city.

Mean daily concentration was determined by dividing the total concentration by the number of hours of tube exposure. NO₂ concentration is reported as parts per billion (1 μ g/m³ = 0.53 ppb).

Statistical analysis

Descriptive analyses were done for the variables under the study. Individual pollution exposure was estimated by using information on the concentrations of NO_2 in each industrial kitchen together with information on activity time patterns. The number of years of cooking in the same kitchen was multiplied by the kitchen-specific NO_2 concentration and used as an indicator of personal exposure.

Correlations between exposure-time indicator and lung function were estimated using Pearson correlation coefficients (15).

Linear regression models (15) were used to assess the effects of the time-exposure indicator on lung function. Results are reported as percent variation of lung function in terms of predicted FEV_1 and FEF_{25-75} for each ten years of cooking work. Linear regression models were applied to the entire group (total) and to non-asthmatic subjects. In the total group we controlled for smoking status and asthma, while in the non-asthmatic group we controlled for smoking status.

The review board for human studies at hospital 2 approved the protocols and the others adopted its decision. Written informed consent was obtained from all subjects.

Results

Eighty-four percent of the people enrolled in the study were female and most were white and non-smokers (Table 1). All cooks were adults ranging in age from 25 to 64 years. Time of work as a cook ranged from 20 days to more than 30 years and the mean time was almost 10 years (Table 2). Socioeconomic status, based on income and years of study, was similar for all study participants.

Predicted FEV_1 values showed a wide range (Table 2). More than 35% of the cooks presented values equal to or higher than 100% and only 13.5% of them showed values below 80% of Knudson predicted value.

Predicted values of $\text{FEF}_{25.75}$ were smaller than those observed for FEV_1 . The mean value was smaller than 80% (Table 2) and 30% of cooks presented predicted values below 60%.

The percentage of participants with predicted FVC values lower than the normal standard (higher than 80%) was smaller than those observed for the other parameters (Table 2).

In the four hospitals, NO_2 concentrations were higher indoors than outdoors (Figure 1). In three of them, indoor concentrations

Table 1. Characteristics of the study participants.					
Characteristics	N (%)				
Gender					
Female	31 (83.8%)				
Male	6 (16.2%)				
Race					
White	28 (75.7%)				
Non-white	9 (24.3%)				
Smoking habit					
Non-smokers	26 (70.3%)				
Smokers	11 (29.7%)				

Data are reported as number of participants with percent in parentheses.

Table 2. Descriptive analyses of the variables used in the study.

Variable Minimum Maximum Mean \pm SE Age (years) 25.00 64.00 45.30 \pm 10.7 Time as a cook (years) 0.05 32.00 9.85 \pm 9.00 Time-exposure ^a 0.75 2475.50 551.35 \pm 687 FEV1 (L) 1.38 3.69 2.51 \pm 0.56 Predicted FEV1 (%) 59.00 123.00 95.43 \pm 14.5 FEF ₂₅₋₇₅ (L/s) 1.05 3.63 2.24 \pm 0.74 Predicted FEF ₂₅₋₇₅ (%) 25.00 108.00 72.14 \pm 19.4 FVC (L) 1.80 4.60 3.28 \pm 0.74 Predicted FVC (%) 75.00 129.00 103.65 \pm 14.7				
Age (years)25.00 64.00 45.30 ± 10.7 Time as a cook (years) 0.05 32.00 9.85 ± 9.00 Time-exposure ^a 0.75 2475.50 551.35 ± 687 FEV1 (L) 1.38 3.69 2.51 ± 0.58 Predicted FEV1 (%) 59.00 123.00 95.43 ± 14.8 FEF ₂₅₋₇₅ (L/s) 1.05 3.63 2.24 ± 0.74 Predicted FEF ₂₅₋₇₅ (%) 25.00 108.00 72.14 ± 19.4 FVC (L) 1.80 4.60 3.28 ± 0.74 Predicted FVC (%) 75.00 129.00 103.65 ± 14.7	Variable	Minimum	Maximum	Mean ± SD
$\begin{array}{c ccccc} \mbox{Time as a cook (years)} & 0.05 & 32.00 & 9.85 \pm 9.06 \\ \mbox{Time-exposure}^a & 0.75 & 2475.50 & 551.35 \pm 687 \\ \mbox{FeV}_1 (L) & 1.38 & 3.69 & 2.51 \pm 0.56 \\ \mbox{Predicted FEV}_1 (\%) & 59.00 & 123.00 & 95.43 \pm 14.5 \\ \mbox{FEF}_{25.75} (L/s) & 1.05 & 3.63 & 2.24 \pm 0.74 \\ \mbox{Predicted FEF}_{25.75} (\%) & 25.00 & 108.00 & 72.14 \pm 19.4 \\ \mbox{FVC (L)} & 1.80 & 4.60 & 3.28 \pm 0.74 \\ \mbox{Predicted FVC (\%)} & 75.00 & 129.00 & 103.65 \pm 14.5 \\ \end{array}$	Age (years)	25.00	64.00	45.30 ± 10.77
$\begin{array}{c ccccc} \mbox{Time-exposure}^a & 0.75 & 2475.50 & 551.35 \pm 687 \\ \mbox{FEV}_1 (L) & 1.38 & 3.69 & 2.51 \pm 0.54 \\ \mbox{Predicted FEV}_1 (\%) & 59.00 & 123.00 & 95.43 \pm 14.5 \\ \mbox{FEF}_{25.75} (L/s) & 1.05 & 3.63 & 2.24 \pm 0.74 \\ \mbox{Predicted FEF}_{25.75} (\%) & 25.00 & 108.00 & 72.14 \pm 19.4 \\ \mbox{FVC} (L) & 1.80 & 4.60 & 3.28 \pm 0.74 \\ \mbox{Predicted FVC} (\%) & 75.00 & 129.00 & 103.65 \pm 14.5 \\ \end{array}$	Time as a cook (years)	0.05	32.00	9.85 ± 9.06
$ \begin{array}{c ccccc} {\sf FEV}_1 \ (L) & 1.38 & 3.69 & 2.51 \pm 0.54 \\ {\sf Predicted} \ {\sf FEV}_1 \ (\%) & 59.00 & 123.00 & 95.43 \pm 14.5 \\ {\sf FEF}_{25.75} \ (L/s) & 1.05 & 3.63 & 2.24 \pm 0.74 \\ {\sf Predicted} \ {\sf FEF}_{25.75} \ (\%) & 25.00 & 108.00 & 72.14 \pm 19.4 \\ {\sf FVC} \ (L) & 1.80 & 4.60 & 3.28 \pm 0.74 \\ {\sf Predicted} \ {\sf FVC} \ (\%) & 75.00 & 129.00 & 103.65 \pm 14.5 \\ \end{array} $	Time-exposure ^a	0.75	2475.50	551.35 ± 687.59
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	FEV ₁ (L)	1.38	3.69	2.51 ± 0.58
$\begin{array}{cccc} FEF_{25\text{-}75} \ (\text{L/s}) & 1.05 & 3.63 & 2.24 \pm 0.74 \\ Predicted \ FEF_{25\text{-}75} \ (\%) & 25.00 & 108.00 & 72.14 \pm 19.4 \\ FVC \ (\text{L}) & 1.80 & 4.60 & 3.28 \pm 0.74 \\ Predicted \ FVC \ (\%) & 75.00 & 129.00 & 103.65 \pm 14.5 \\ \end{array}$	Predicted FEV ₁ (%)	59.00	123.00	95.43 ± 14.54
$\begin{array}{c cccc} \mbox{Predicted FEF}_{25.75} \ (\%) & 25.00 & 108.00 & 72.14 \pm 19.4 \\ \mbox{FVC (L)} & 1.80 & 4.60 & 3.28 \pm 0.74 \\ \mbox{Predicted FVC (\%)} & 75.00 & 129.00 & 103.65 \pm 14.5 \\ \end{array}$	FEF ₂₅₋₇₅ (L/s)	1.05	3.63	2.24 ± 0.74
FVC (L) 1.80 4.60 3.28 ± 0.74 Predicted FVC (%) 75.00 129.00 103.65 ± 14.15	Predicted FEF ₂₅₋₇₅ (%)	25.00	108.00	72.14 ± 19.42
Predicted FVC (%) 75.00 129.00 103.65 ± 14.	FVC (L)	1.80	4.60	3.28 ± 0.74
	Predicted FVC (%)	75.00	129.00	103.65 ± 14.15

Data are reported as minimum and maximum values and as means \pm SD for 37 participants.

^aTime of work as a cook (years) multiplied by NO₂ concentration (ppb). FEV₁ = forced expiratory volume in 1 s; FEF₂₅₋₇₅ = forced expiratory flow between 25 and 75% of FVC; FVC = forced vital capacity.

were higher than 60 ppb. In hospitals 2 and 4, NO₂ levels inside and outside the kitchen were very close, although inside pollutant levels in kitchen 4 were 4-fold higher than in kitchen 2. At the same time, the NO₂ concentration at the central bus station was 28 ppb and the mean value for the 4 monitoring points was 11.46 ppb.

Table 3 presents the effect of the time/ exposure indicator on the predicted FEV₁ and FEF₂₅₋₇₅ values for the professional cooks included in the present study. A decrease of predicted FEV_1 related to the time/exposure variable was observed in both groups. On average, for the cooks analyzed in this study, each year of work as a cook corresponded to decreases in predicted FEV₁ ranging from 2.3% for non-asthmatic subjects to 2.5% for the group as a whole. When smoking status and asthma were included in the analysis the effect of time/exposure decreased about 10% and lost statistical significance. On the other hand, the effect of time/exposure on predicted FEF₂₅₋₇₅ was more robust than that observed for FEV₁. The inclusion of controllers for smoking status and asthma did not affect the effects of time/exposure on pulmonary function parameter. In our sample, each year of work as a professional cook corresponded to a 3.6% decrease of predicted FEF₂₅₋₇₅ values.

It is possible to estimate that after a 10year period of work as cooks the participants of the study may present decreases in both predicted FEV₁ and FEF₂₅₋₇₅ that can reach 20 and 30%, respectively.

Our analysis did not show any statistically significant effect of NO_2 exposure on FVC values.

Discussion

Changes in lung function of cooks induced by gas stove exposure have been under-explored. The present study is the first one to focus on the potential adverse effects of indoor exposure to gas stove-generated NO_2 on the lung function of professional cooks. The results of the present study indicate that decreases in FEV_1 and FEF_{25-75} may correlate with time of work as a cook and with NO_2 concentration in the work place. Also, it is possible to estimate that ten years of work as a cook may result in 20 and 30% decreases in the predicted values of these two lung function indicators, respectively.

This cross-sectional study used data for all 37 professional cooks that were working in the 4 largest hospital kitchens in Araraquara at the time of the study. Information on lung function and quasi-individual exposure was measured by standard procedures and was not biased by misclassification.

We used the same kind of kitchen, i.e., hospital kitchen, to avoid differences in food and cooking methods. Svendsen et al. (16) suggested that in restaurant kitchens there are other aerosols generated from frying vegetables and animal fats that can be harmful to the lungs. Ng et al. (11), in a study on 1438 non-professional female cooks, suggested that cooking oil mists, animal fats, and other thermal products of cooking may irritate the mucous membrane when the food is being prepared. Hospital food is usually poor in frying and in fat acids. This is an important factor to be controlled because there are many elements other than NO_2 that can be present in kitchen aerosols and may induce damage to the lungs. In addition to aerosols, cooking with gas may be associated with some other environmental factors, which of themselves may be risk factors for respiratory illness. The concentration of NO₂ is well correlated with the concentration of carbon monoxide, particulate polycyclic aromatic hydrocarbons and soot. For this reason, NO2 measurement is an important indicator of indoor air pollution (17). Since we did not measure other kitchen air contaminants we decided to use NO2 measurements as a proxy for kitchen air pollution.

Palmes' (14) diffusion tube only provides information on average exposure for a given period of time and does not take into account intermittent peak exposures to NO₂. Harlos and Spengler (18), using an extensive data set on NO₂ exposure, reported that while 1-h maximum personal exposure for cooks can reach around 180 ppb, 5-s peaks of 500 ppb were observed. In our study we were not able to measure peaks of NO₂ concentration and therefore we could not estimate the acute effects of the gas. Despite this weakness, the approach we used to estimate exposure is much more reasonable than using dichotomous variables to define exposure to NO₂ (presence of gas stove - yes or no).

The indoor concentration of NO_2 and other pollutants depends on the indoor



Figure 1. NO_2 concentrations inside and outside the hospital kitchens analyzed in the present study.

Table 3. Regression coefficients, standard error of the means, and statistical significance of linear regression models estimating the effect of the NO₂ time/exposure indicator on predicted FEV₁ and FEF₂₅₋₇₅ in a single model and in models controlling for smoke status and for both smoke and asthma status.

Model To				Non-asthmatics		
	Coefficient	SEM	Р	Coefficient	SEM	Ρ
FEV ₁						
TEI (single)	-2.507	1.212	0.046*	-2.288	1.145	0.054
TEI (smoke)	-2.296	1.253	0.076	-2.089	1.186	0.088
TEI (smoke + asthma)	-2.299	1.231	0.071			
FEF ₂₅₋₇₅						
TEI (single)	-3.525	1.608	0.035*	-3.351	1.483	0.031*
TEI (smoke)	-3.625	1.674	0.037*	-3.670	1.529	0.023*
TEI (smoke + asthma)	-3.631	1.614	0.031*			

 FEV_1 = forced expiratory volume in 1 s; FEF_{25-75} = forced expiratory flow between 25 and 75% of FVC; TEI = time/exposure index.

*Statistical significance (t-test).

sources, the conversion to other compounds, and removal by ventilation. Personal exposure can be influenced by time-activity patterns. Differences in NO2 concentrations among the kitchens may be attributed to different kitchen sizes and ventilation patterns. Adopting present levels of NO₂ exposure as representative of the entire work period of each professional may be controversial. However, the areas of each kitchen and their physical structure, as well as the number of beds in the four hospitals, did not change substantially since they were built. Thus, we assumed that NO₂ levels remained almost the same since the beginning of the exposure period.

It has been suggested that the effect of gas stove emissions on respiratory symptoms can be modified by socioeconomic status (19). The cooks included in our study had the same income and educational level, so that modification of the effects by socioeconomic status was less likely.

A passive NO₂ sample provides a mean estimate of ambient concentrations. Hence, it does not allow analysis of the changes in pollutant concentrations during the period of exposure. Studies carried out in houses with gas stoves showed that the highest concentrations were found in kitchens and that people did not spend most of their time in the kitchen (12,20). In our study, the indoor concentrations of NO₂ in each hospital kitchen were higher than the ambient hospital level, in agreement with the concentrations observed in the house studies. However, in contrast to home cooks, professional cooks are continuously exposed to NO₂ in the kitchens throughout the working period (6 h a day, in general).

The association between indoor NO_2 exposure and respiratory symptoms or decreased lung function has been a matter of discussion. The most frequently studied endpoints in investigations of the health effects of indoor NO_2 are respiratory illness and/or symptoms in children (21-23), adolescents

(24), and adults (11,12,16,25), while others observed no such effects in infants and schoolchildren (6,26), adolescents (27) or adults (28-30). Some investigators found effects in girls but not in boys (31) and in women but not in men (12). Most of the discrepancies in the results of these studies may be attributed to misclassification of exposure and/or outcome information.

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FEV₁ and FEF₂₅₋₇₅, lung function parameters that are used to evaluate obstructive patterns, were used in the present study to evaluate inflammatory effects on small airways. The main site of NO₂ deposition and injury is in the distal conducting airways at the level of the terminal bronchioles (32). Kim and Kang (33) suggest that NO₂ induces small obstructive changes in peripheral airways and Blomberg et al. (34) showed that in healthy subjects the major site of inflammation following exposure to NO₂ may be in the smaller airways and not in the alveoli. In the present study, changes occurred in both FEV1 and FEF25-75 supporting the hypothesis that NO₂ effects are more relevant in very small airways. The lack of effects on FVC parameters can be attributed to their poor capacity to evaluate obstructive lung diseases.

Experimental studies on healthy nonsmoking subjects have shown that adverse inflammatory effects on the airways may occur due to acute exposure to NO_2 (2 ppm) and can be determined by bronchoalveolar lavage (35) and FEV₁ measurements (36). Frampton et al. (35) found an inflammatory response that persisted at least 18 h after exposure, while Blomberg et al. (36) demonstrated major decrements in FEV₁ just after exposure and this lung function indicator response was attenuated after repeated exposures, even in the presence of a persistent neutrophilic inflammation in the airways. They concluded that NO₂ is a proinflammatory air pollutant under conditions of repeated exposure. Measurements made in the vicinity of gas stoves have reported NO₂ levels ranging from 0.3 (18) to 0.6 ppm (37) during operating periods of time that may persist for 20 to 60 min after the cook has stopped working, and peaks that may exceed 2 ppm (38).

In our study, we supposed that cooks were exposed to at least two peaks of NO_2 concentrations a day for many years. In addition, the presence of indoor pollutants, even at low concentrations, may have an important biological impact because of long periods of exposure. This is much more than the four peaks reported in experimental study (36). In this real situation, more than producing a chronic and silent inflammatory process, NO_2 and the other pollutants can really be harmful, producing chronic damage to the respiratory system.

The present study showed a statistically significant but small adverse effect of gas stove exposure on lung function of professional cooks that was related to the length of working in the same position and to the concentration of NO_2 . Different study designs must be implemented in order to confirm this apparent causal relationship. Also, these results support the recommendation that alternative and improved methods of cooking besides appropriate ventilation of all indoor combustion appliances, including gas stoves, should be adopted in industrial kitchens.

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