Doença pulmonar obstrutiva crônica em mulheres expostas à fumaça de fogão à lenha

Palavras-chave:
Doença pulmonar obstrutiva crônica
Biomassa
Fumaça

RESUMO

Objetivo: Identificar sintomas respiratórios e DPOC (relação entre volume expiratório forçado no primeiro segundo e capacidade vital forçada < 0,70 e abaixo do limite inferior da normalidade) em mulheres não fumantes, com história de exposição à fumaça da combustão de lenha de ao menos 80 horas-ano.

Métodos: Foram incluídas 160 mulheres não tabagistas. Coletaram-se dados demográficos, sintomas e informações sobre outras exposições ambientais. Todas as mulheres realizaram espirometria e aquelas com DPOC também medidas de volumes pulmonares.

Resultados: O grupo com DPOC apresentava maior duração de exposição, em anos, à fumaça de lenha (p = 0,043), maior tempo de domicílio rural (p = 0,042), duração similar de tabagismo passivo (p = 0,297) e de trabalho na lavoura (p = 0,985). Tosse (69,8%), expectoração (55,8%) e chiado (67,4%) predominaram no grupo com DPOC (p < 0,001) quando comparado ao grupo sem DPOC (40,2%, 27,4%, 33,3%, respectivamente). As pacientes com DPOC apresentavam distúrbio obstrutivo leve a moderado e volumes pulmonares normais, exceto a relação entre o volume residual e a capacidade pulmonar total (VR/CPT) > 0,40 em 45%, que apresentou correlação negativa com o VEF1 e VEF1/CVF.

Conclusão: Mulheres com exposição prolongada à fumaça de lenha apresentaram DPOC predominantemente leve a moderado. Aquelas sem DPOC tiveram alta prevalência de sintomas respiratórios crônicos, justificando monitoramento clínico e espirométrico.

Introduction

In developing countries, biomass fuels (wood, charcoal, animal dung and crop residues) are used for space heating and for cooking food in rustic stoves. In Brazil, the Brazilian Institute of Geography and Statistics[1] has estimated at 40.9% the proportion of rural residents and 2.6% the proportion of urban dwellers who use wood stoves.

Approximately 1.5-2 million deaths per year worldwide are attributed to diseases related to pollution from biomass combustion.[2] Reviews and meta-analyses have demonstrated that exposure to biomass smoke is an important risk factor for chronic obstructive pulmonary disease[2-4] (COPD). Approximately 3 billion people worldwide are exposed to biomass smoke, whereas the number of smokers is much lower, 1.1 billion, thus making exposure to biomass smoke a major risk factor for COPD, globally.[5] However, the findings of studies from other countries may not be fully applicable to Brazil, as the vegetation used as fuel here is different, the use of biomass is mostly restricted to cooking food and there is limited use of other types of biomass.

This study aims to identify respiratory symptoms and COPD in non-smoking women with a history of exposure to wood stove smoke for at least 80 hours-year.

Methods

The participants were recruited from outpatient clinics of a university hospital and two Basic Health Units.

Inclusion criteria were age ≥ 40 years, never having been a smoker, exposure to wood smoke while cooking for ≥ 80 hours/year and a minimum of 10 years[6]; signing the free and informed consent form. Exclusion criteria were: presence of clinical evaluation by pulmonologists performed during the study or contained in medical records, suggesting bronchial asthma and/or allergic rhinitis; extrapulmonary disease that could interfere with lung function; change in forced expiratory volume in one second (FEV_1) after bronchodilators (bd) ≥ 10% in relation to the predicted value.[7]

The adapted version of the questionnaire used in the PLATINUM study in Brazil was used.[8] Data were obtained regarding demographics, respiratory symptoms (cough, phlegm, wheezing and dyspnea); previous medical diagnosis of COPD, exposure to smoke resulting from wood combustion (intensity, presence of stove chimney, location in the house) and other types of exposure (passive smoking, agricultural activity).

Exposure to wood stove smoke was expressed in hours, years and hours-year (product of time in years cooking with wood stove, multiplied by the mean number of hours spent in this activity).[6] Passive exposure to tobacco was expressed in years of living in a home with smokers.

All women interviewed underwent spirometry pre- and post-bd use. The following parameters were measured: forced vital capacity (FVC), FEV_1; ratio between FEV_1 and FVC. Women who were diagnosed with COPD also underwent residual volume (RV) and total lung capacity (TLC) assessment. Pulse oximetry (SpO_2) at rest was measured in women with FEV_1 < 50% of the predicted value.[9]

The diagnostic criteria for COPD were: history of dyspnea and/ or chronic cough; absolute value of FEV_1/FVC

Table 1 – Demographic profile of women exposed to wood smoke.

Variables[a]	Group with COPD (n = 43)	Group without COPD (n = 117)	*p* value
Demographic data			
Age (years)	70.8 (8.4)	64.2 (10.7)	< 0.001[b]
40-50	0 (0.0)	16 (13.7)	–
51-60	5 (11.6)	42 (35.9)	–
61-70	15 (34.9)	3 (29.1)	–
71 or older	23 (53.5)	25 (21.4)	< 0.001[c]
BMI (kg/m²)	25.9 (6.2)	28.6 (6.6)	0.021
Self-reported ethnicity (n, %)			
Mulatto + Black	23 (53.5)	57 (48.7)	–
Caucasian	20 (46.5)	58 (49.6)	0.755
Native Brazilian	0 (0.0)	2 (1.7)	–
Schooling (years)	2.4 (2.4)	3.1 (2.9)	0.211
Rural residence (years)	39.9 (18.1)	33.7 (16.6)	0.042 [d]

BMI, body mass index; COPD, chronic obstructive pulmonary disease.
[a] Data expressed as n and % or mean and standard deviation.
[b] OR = 1.10 (95%CI: 1.05-1.14).
[c] OR = 2.91 (95%CI: 1.82 - 4.65).
[d] OR = 1.02 (95%CI: 1.00-1.04).

post-bronchodilator use < 0.70,[7,10] and below the lower limit of normality[11] (LLN) and exclusion of other diseases with symptoms and spirometric alterations similar to those found in COPD: tuberculosis, pneumoconiosis, cystic fibrosis, bronchiolitis and alpha-1 antitrypsin deficiency.

The classification of COPD severity was based on FEV_1 as a percentage of the predicted value: mild disease – $FEV_1 \geq 80\%$; moderate disease – $FEV_1 < 80\%$ and $> 50\%$; severe disease – $FEV_1 < 50\%$ and $> 30\%$; very severe disease – $FEV_1 < 30\%$ or $< 50\%$ with signs of chronic respiratory failure[9] (defined by the presence of arterial oxygen pressure (PaO_2) < 60 mmHg). In this study, oxygen saturation by pulse oximetry was defined at < 92%.

The lung function tests, pre- and post-bd use were performed and interpreted according to the guidelines of the Brazilian Society of Pneumology and Tisiology (SBPT) and the American Thoracic Society[7,12,13] (ATS). The predicted values used were those of Knudson[7] (1983). Normal limits for TLC were: 60-120% of predicted; for RV: 60-140% of predicted, and for the ratio between residual volume and total lung capacity, RV / TLC < 0.40.[7]

A Jaeger® Masterscreen spirometer, calibrated daily and recalibrated when necessary was used in the study and for the spirometries performed at the Basic Health Units, an EasyOne, NDD Medizintechnik spirometer was used, of which calibration was verified daily.

Measurements of lung volumes were made by helium gas dilution technique in a closed circuit and with multiple breaths.[7,13] Pulse oximetry was performed using a portable Dixtal® DX 2405 oximeter.

The body mass index (BMI) was calculated using the formula: $BMI = W/H^2$, (W = weight in kg and H^2 = the square of height in meters). BMI values between 18.5-24.9 m^2/kg were considered normal.[14]

The statistical analysis was performed with the software SPSS® release 15.0 for Windows®. Student's *t* test was used for comparison of groups regarding quantitative variables with normal distribution or Mann-Whitney U test, for the non-normal distribution. The Chi-square and Fisher's exact tests were used to compare frequencies of qualitative variables and calculate the risk that the independent variable represented. Pearson's or Spearman's correlation coefficient was calculated to analyze the correlation between lung function tests, and between them and the duration of exposure to risk factors, respectively, for the normal and non-normal distribution data. The level of significance was set at $p < 0.05$ and odds ratio (OR) with 95% confidence interval (95% CI) were calculated.

This study was approved by the local Ethics Committee, on 04/04/2008, under Registration N. 018/08.

Results

Of the 160 assessed women, 43 (26%) met the criteria for the diagnosis of COPD, whereas 117 did not. The COPD group had an older mean age, consisting predominantly of women older than 70 years ($p < 0.001$), significantly lower BMI, on average, but slightly above the upper limit of normality. The two groups were similar regarding self-reported ethnicity and level of schooling. The COPD group had lived longer in rural areas (Table 1).

The duration of exposure to farming activities in years, in the COPD group, was similar to that without COPD (mean 17.7 ± 15.0 and 17.7 ± 14.5, respectively, $p = 0.985$). The work was intermittent, alternating with housework, especially cooking food. The exposure was mostly to grain dust (rice, beans, corn and cotton) and pesticides.

The group with COPD had greater duration in years of exposure to smoke from wood combustion. Most women had length of exposure of more than 20 years. There was no

Table 2 – Characteristics of exposure in both groups.

Variables[a]	Group with COPD (n = 43)	Group without COPD (n = 117)	*p* value
Exposure to wood smoke			
Hours per day (mean + SD)	6.1 (1.9)	6.2 (1.9)	0.760
Duration (years-mean + SD)	35.0 (12.3)	30.5 (12.2)	0.045[b]
10-20 years (n, %)	7 (16.3)	35 (29.9)	–
21-30 years	13 (30.2)	33 (28.2)	–
31-40 years	8 (18.6)	30 (25.6)	0.022[c]
41 years and longer	15 (34.9)	18 (15.4)	–
Total	43 (100.0)	117 (100.0)	–
Cumulative (h-years-(mean + SD)	211.2 (98.2)	189.9 (104.5)	0.248
80-120 h-year (n, %)	11 (25.6)	40 (35.0)	–
121-160 h-year	7 (16.3)	20 (17.1)	0.181
161-200 h-year	4 (9.3)	15 (12.8)	–
201 h-year or >	21 (48.8)	42 (35.0)	–
Total	43 (100.0)	117 (100.0)	–
Stove inside the house (n, %)	37 (86.0)	102 (87.2)	0.850
Stove without chimney	17 (30.95)	49 (49.0)	0.798
Passive smoking (years)	29.7 (21.0)	26.0 (19.6)	0.297
Straw cigarettes (n, %)	26 (72.2)	62.0 (68.1)	–
Manufactured cigarettes	10 (27.8)	29 (31.9)	0.652

COPD, chronic obstructive pulmonary disease; SD, standard deviation.
[a] Data expressed in n and % or in mean and standard deviation.
[b] OR = 1.03 (95% CI 1.0007-1.06).
[c] OR = 1.47 (95% CI 1.06-2.05).

difference between the two groups regarding the location of the stove out of the house and the presence of a stove chimney, factors that decrease the intensity of exposure. Women often pointed out that the kitchen walls were blackened due to smoke. The stove, when located outside the home, was usually kept in a porch located next to the kitchen. There was no significant difference in the type of cigarette associated with passive exposure and duration of passive smoking (Table 2).

Symptoms such as chronic cough, phlegm and wheezing, prevalent in both groups, were significantly more frequent in the COPD group. The risk for these symptoms is three to four times higher when compared to the group without COPD (Table 3). The complaint of dyspnea occurred in similar proportions in both groups; the mean scores of the Medical Research Council (MRC) were similar between groups. Only three patients from the COPD group had a score of 4.

Women with COPD had FVC, FEV_1 and FEV_1/FVC ratio consistent with obstructive lung disease, predominantly mild to moderate in severity (Table 3). None of the women had very severe COPD, or complications of COPD, such as cor pulmonale. All women with $FEV_1 < 50\%$ showed oxygen saturation > 92% in the oximetry at rest.

There was no correlation between the magnitude of the obstruction, assessed by FEV_1, FEV_1/FVC and RV/TLC against age, intensity of exposure (in hours, years and hours-years) and passive smoking. There was no association between spirometric parameters and the presence of cough, expectoration, and dyspnea. The group of patients who complained of wheezing had significantly lower FEV_1 than those who did not have the symptom ($p < 0.04$).

All women with COPD had TLC and RV within normal limits. The RV/TLC ratio was above the upper limit of normality in 45% of patients. The greater the values of RV/TLC ratio, the smaller the values of FEV_1 and FEV_1/FVC ratio ($p = 0.001$, $r = –0.721$ and $p = 0.006$, $r = –0.435$, respectively).

Discussion

This study aimed to evaluate the possibility of COPD diagnosis in non-smoking women exposed to wood smoke for at least 10 years and 80 hours/year. To minimize the effect of decreased FEV_1/FVC due to the influence of age, FEV_1/FVC ratio < 0.70, and lower than the lower limit of normality were used as criteria for the diagnosis of COPD.[8,10,11] Of the 160 women studied, 43 had symptoms and lung function compatible with COPD. On average, they were older, had longer exposure to firewood smoke in years, had respiratory symptoms more frequently and had lived longer in the rural area.

The COPD group in this study had older mean age than the group without COPD, and although the disease was predominant in women older than 70 years, it was also diagnosed in younger individuals. Similar age distribution was previously reported in a patient with COPD due to exposure to wood smoke.[15,16] A study of prevalence of COPD developed in Brazil[8] showed that COPD increased with age, with the highest prevalence after the age of 60.

In this study, the COPD group showed longer duration in years of exposure to wood smoke and longer time of residence in the rural area. Older women were less affected

Table 3 – Respiratory symptoms and lung function in women exposed to wood smoke.

Variables[a]	Group with COPD (n = 43)	Group without COPD (n = 117)	*p* value
Chronic cough (n, %)	30 (69.8)	47 (40.2)	0.001[d]
Expectoration	24 (55.8)	32 (27.4)	0.001[e]
Wheezing	29 (67.4)	39 (33.3)	0.001[f]
Dyspnea	35 (81.4)	86 (73.5)	0.303
FVC (% of predicted)	95.2 (15.2)	104.2 (15.8)	0.002
FEV_1 (% of predicted)	68.8 (17.2)	100.8 (15.6)	< 0.001
FEV_1/FVC	0.57 (0.1)	0.79 (0.1)	0.029
FEV_1/FVC LLN	0.69 (2.4)	0.70 (2.9)	–
TLC (% of predicted)[b]	90.2 (10.9)	–	–
RV (% of predicted)[b]	89.2 (18.6)	–	–
RV/TLC[b]	0.44 (0.07)	–	–
RV/TLC > 0.40 (n, %)	19 (45.0)	–	–
COPD staging (n, %)			
Mild	9 (21.4)	–	–
Moderate	26 (61.9)	–	–
Severe	7 (16.7)	–	–
Very severe	0 (0.0)	–	–

COPD, chronic obstructive pulmonary disease; FVC, forced vital capacity; FEV_1, forced expiratory volume in the first second; FEV_1/FVC, FEV_1/forced vital capacity ratio; LLN, lower limit of normality; TLC, total lung capacity; RV, residual volume; RV/TLC, RV/TLC ratio.
[a] Data expressed as n and % or mean and standard deviation.
[b] n = 38.
[c] n = 46.
[d] OR = 3.44 (95%CI: 1.63-7.27).
[e] OR = 3.36 (95%CI: 1.62-6.94).
[f] OR = 4.14 (95%CI: 1.96-8.72).

by the Brazilian rural exodus that has occurred intensely and progressively in the last four decades.[17] Having lived longer in the rural areas, they have been exposed to wood stove smoke for longer periods of time. It was observed in this study that exposure was, on average, 200 hours-year in the COPD group. In a Mexican study, women with a similar level of exposure showed a 10-fold higher risk for chronic bronchitis.[18] The highest risk for COPD, reported in a meta-analysis[4] was exposures greater than 200 hours-year, for over 30 years and more than 4 hours/day, values similar to those described in the present study.

The burning of biomass can generate during the use of stoves mean concentrations of about 30,000 mg/m^3, above the safety standards set by WHO[2] (150 g/m^3). A study carried out in the Brazilian northeast region quantified the level of soot ("black carbon") and particulate matter ($PM_{2.5}$) in households that used wood stoves. A correlation was obtained between pollution intensity and occurrence of individuals with chronic respiratory symptoms and lung function impairment. The prevalence of COPD in the study group was 20%, similar to that found in the present study.[19] This shows again that the use of biomass for cooking is the main source of indoor air pollution and that wood generates higher mean concentration of PM_{10} (respirable particulate matter with a diameter <10 micrometers) than charcoal.[2]

The reporting that the kitchen walls were blackened suggests inadequate chimney use and high level of pollution. The percentage of reported location of the stove outside the house did not differ between the two groups, suggesting that this was not a strong factor regarding the degree of pollution and risk for COPD. Airtight stoves produce lower intensity pollution, but still above the tolerable.[2] It has been shown that improving the quality of the wood stoves decreases the morbidity related to the use of biomass.[20]

A study on the effects of passive smoking suggests the possibility that the exposed individual can have bronchial remodeling after repeated exposures.[21] Another study showed an association between passive smoking and COPD in household exposures ≥ 42 years[22] (OR = 1.68, 95%CI: 1.19 to 2.38). The mean duration of exposure to passive smoking in the present study was below the level considered of greater impact in the abovementioned study, and there was no significant difference between groups. This exposure was probably not a relevant factor for COPD onset, even with the prevalence of exposure to straw cigarette smoke, which would lead to an additional risk.[23] In Southern Brazil, a study with 1479 farmers showed OR for respiratory symptoms, adjusted for smoking, of 1.30 (95%CI: 0.67 to 1.33) due to exposure to high levels of grain dust.[24] This risk rate is lower than that caused by exposure to burning biomass[3,4] (OR: 2.4-2.8). In the present study, exposure to agricultural products, due to its intermittent characteristic, low duration of exposure and diversity of products, seems to have been unimportant in the development of COPD; the duration of exposure was similar in both groups.

It is noteworthy, in the present study, the complaint of chronic cough in 40% of women without COPD. In those with

lung function close to the lower limit of normality, there is the possibility of an established bronchial inflammation, although lung function is not compatible with COPD. In smokers followed for five years, there was an increase of 7.5% to 24.8% in the percentage of individuals who had bronchial obstruction.[25] A recent review article on obstructive lung diseases related to exposure to biomass suggests these longitudinal studies.[26] Dyspnea was, in the present study, the most prevalent symptom in both groups, with no significant difference between them, probably due to the high subjectivity of this symptom. In a 2010 study, the risk of woodstove users presenting respiratory symptoms was nearly four-fold higher for cough associated with expectoration when compared to unexposed individuals.[27] In this study, if the diagnostic criterion of COPD had included only respiratory symptoms, there probably would have been underdiagnosis and overdiagnosis. A 2004 study described a 43% increase in the diagnosis of COPD when using spirometry, rather than just a symptom questionnaire.[28]

The absence in the present study, of an association between FEV_1 and FEV_1/FVC with cough and expectoration symptoms. is possibly related to the low prevalence of severe COPD, as the frequency of symptoms increases with disease severity.[29]

The predominant pattern of mild to moderate obstructive disease, reported in this study, has been described before[2,16] and also in a Brazilian study.[30] Compared with changes related to COPD due to tobacco use, those due to exposure to biomass smoke are less pronounced.[30] There have been reports of severe COPD associated with biomass smoke in countries which, differently from Brazil, use animal dung and crop residues as fuel, not only for cooking, but also for heating the environment.[31] In our study, increased levels in the RV/TLC ratio were associated with lower FEV_1 and FEV_1/FVC, suggesting air trapping caused by obstruction to airflow. Endoscopic and histopathological studies in women exposed to smoke from wood combustion showed that bronchial changes predominate, with frequent findings of anthracotic endobronchitis, small airway fibrosis[31] and minimum areas of emphysema.[16]

We found no association between spirometric alterations and intensity and duration of exposure. The reasons were possibly the sample size and/or not using an objective method to measure exposure. However, the COPD group had a significantly longer exposure than the group without COPD, indirectly suggesting this association, which has been previously reported.[15]

It is noteworthy the fact that a substantial number of women exposed to wood smoke did not have the disease. This is a well known fact among smokers as well; only 25-40% of them do not develop COPD.[9] Individual factors, possibly genetically-mediated ones, could explain this fact.[9]

A possible limitation of this study was the fact that the data were collected by questionnaire. However, this questionnaire has been used in Brazil, in the PLATINUM Study, and the interviewers were trained to use it. To assess the duration and intensity of exposure, and reduce the possibility of recalling errors, we tried to use as a reference the participants' facts of life that could be easily recalled: age when she started cooking, the year she moved to an urban area and the year she married. Other possible limitations include the lack of objective measurement of the degree of environmental pollution in a sample of houses and the helium gas dilution method to measure lung volume, as this method may underestimate the volumes in the presence of emphysema. On the other hand, it has been reported that patients with COPD associated with biomass smoke do not have emphysema, or have minimum areas of emphysema, so the impact of the measurement technique was probably nonsignificant.[16]

On the other hand, the following showed to be relevant factors in this study: the diagnosis of COPD using two parameters of lung function; the evaluation of data on other environmental exposures (passive smoking and cigarette type, farm work, location and characteristics of the stove), allowing better assessment of the role of wood smoke as an etiological factor of COPD; the exclusion of women smokers; criterion for inclusion in the study that required a minimum duration of exposure to wood smoke.

Conclusion

The present study suggests that older women living in the rural area for a long time and exposed to wood smoke for prolonged periods are at risk for COPD. In these women, spirometry increases the chance of disease diagnosis, even when respiratory symptoms are not very significant. On the other hand, chronic respiratory symptoms in women exposed to wood smoke for a relevant length of time, even with normal spirometry results, presupposes the need for clinical and spirometric follow-up.

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Conflicts of interest

The authors declare no conflicts of interest.

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