

## Original Research Article

# Effect of passive smoking as a risk factor for chronic obstructive pulmonary disease in normal healthy women

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### ABSTRACT

**Background:** Environmental tobacco smoke (ETS) is a risk factor for cardiovascular disease, asthma in children and lung cancer. There is a biological plausibility of ETS as a causal factor for COPD. Objectives of the study were to examine the effect of passive smoking on lung function in non-smoking healthy women and to co-relate the effects of passive smoke as a risk factor for COPD.

**Methods:** 50 women between 20-40 years of age exposed to passive smoke at home and workplace were assessed by questionnaire. The pulmonary function tests were performed and the values of FEV1 and FVC were obtained by a spirometer.

**Results:** Out of 50 women, 34 % at workplace, 54% at home and 12% at home and workplace were exposed. Mean age was 30.3 years. Mean±SD of FEV1 was 1.94±0.9, FVC was 2.54±1.06, FEV1/FVC was 73.5±10.06 predicted FEV1 % was 63.2±23.2. FEV1/FVC of women exposed at home and workplace was 70.84 indicating that they have higher chances of developing COPD later in life.

**Conclusions:** Passive smoking represents a serious health hazard that can be prevented by health education campaigns.

**Keywords:** Environmental tobacco smoke, Pulmonary function tests

## INTRODUCTION

Passive smoking or environmental tobacco smoke (ETS) is a combination of side stream smoke emitted from the burning end of a cigarette and the remainder of mainstream smoke exhaled by a smoker. The side stream smoke constitutes about 85% of the smoke present in the room where active smokers smoke, and contains many potentially toxic components, some of which may occur in even higher concentrations than in the mainstream smoke.<sup>1</sup>

The particulate phase consists of tar, nicotine, benzo(a)pyrene, and hundreds of other noxious compounds. Active smoking remains the main risk factor

for chronic obstructive pulmonary disease (COPD), yet COPD occurs also in never smokers.<sup>2</sup> Exposure to passive smoking has been implicated as a causal factor of cardiovascular disease, asthma in children and lung cancer and has been estimated to cause about 1% of overall global mortality.<sup>3</sup>

In India, smoking is a common habit in both the urban and rural areas in the form of cigarettes, bidis, pipes, cigar, hookah.<sup>4</sup> There is a biological plausibility of ETS as a causal factor for COPD. Although studies have shown associations between exposure to ETS and respiratory symptoms, a clear causal relationship between ETS and COPD has proved more difficult to establish.<sup>5</sup> COPD is a chronic inflammatory disease characterized by

progressive airflow limitation that is not fully reversible and is estimated to affect hundreds of millions worldwide.<sup>6</sup> Environmental conditions such as the overcrowding and the poor ventilation at homes and work places may make the health effects of ETS more pronounced. Exposures to tobacco smoke leads to more frequent use of health care, absence from work, and in most cases hospitalization for respiratory diseases. The female population is a special category that needs attention because exposure to tobacco smoke can also seriously affect fertility and may lead to complications in pregnancy and childbirth.<sup>7</sup>

**Objectives**

- To examine the effect of passive smoking on lung function in non-smoking normal healthy women.
- To co-relate the effects of passive smoke as a risk factor for COPD.

**METHODS**

This was a cross-sectional study in Padmashree Dr. D.Y. Patil Medical College, Pimpri, Pune, Maharashtra, India. It was conducted on 50 healthy women in the age group between 20- 40 years after obtaining the informed consent and ethical committee approval. Passive smoking was assessed from the questionnaire. Participants with at least one smoker in the household were classified as exposed to tobacco smoke in the household.

The study subjects were asked regarding the number of smokers in family, type of tobacco product smoked, approximate number of cigarettes/bidis smoked per day and years of passive exposure to tobacco smoke. Participants with at least one smoker in the room where they worked are classified as passive smokers in the workplace.<sup>8,9</sup> Details of occupational history and duration were taken. Height and weight was performed with subjects wearing light clothing but without shoes. Body mass index (BMI) was calculated by weight/height<sup>2</sup> [kg/m<sup>2</sup>].

Airway narrowing was assessed by asking the patients to blow out as hard and as fast as they can into a spirometer. The values of FEV1 and FVC were obtained by maximal forced expiration into a spirometer. The volume that has been exhaled at the end of the first second of forced expiration (FEV1), measured in liters.

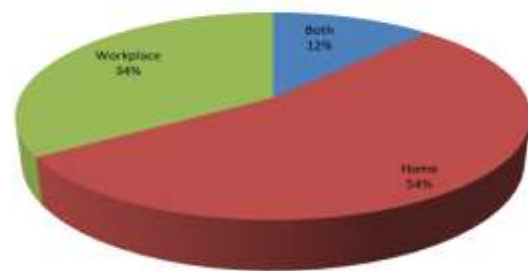
The vital capacity from a maximally forced expiratory effort is called the forced vital capacity (FVC), also measured in liters. COPD is defined using the fixed ratio of definition of FEV1/FVC <0.7 and disease severity staging according to the GOLD (Global Initiative for Chronic Obstructive Lung disease) guidelines.<sup>(10)</sup> Reversibility was tested using 0.8 mg salbutamol in all subjects with either FEV1/FVC <0.7 or FEV1<90% of the predicted value. Statistical analysis using mean, standard deviation and P value was done.

**Exclusion criteria**

Subjects with history of wheezing, sputum production, cough, allergic rhinitis, dyspnea, hay fever, urticaria, chest deformities, allergic conditions, cardiac and respiratory diseases are excluded. Pregnant women were excluded. Women with any occupational exposure to hazardous smoke and using biomass fuel (chulha) for cooking were excluded from the study.

**RESULTS**

Out of 50 women, 34% females at workplace, 54% females at home and 12% at home and workplace were exposed to ETS (Figure 1).

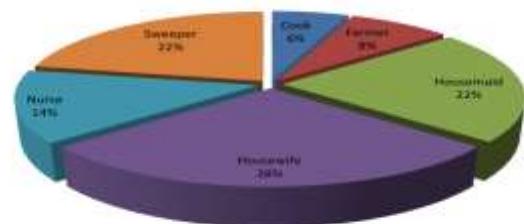


**Figure 1: Place of exposure frequency.**

Mean age is 30.3 years. Mean FEV1 is 1.94±0.9, Mean FVC is 2.54±1.06, Mean FEV1/FVC is 73.5±10.6, Mean Predicted FEV1 is 63.2±23.2 shown in Table 1.

**Table 1: Characteristics of variables with mean and standard deviation.**

Parameter	Mean±SD
Age (years)	30.3±5.4
Height (m)	1.50±0.1
Weight (kg)	47.3±6.1
BMI	21.7±2.1
Pulse	78.9±6.8
FEV1 (litres)	1.94±0.9
FVC (%)	2.54±1.06
FEV1/FVC	73.5±10.6
Predicted FEV1 (%)	63.2±23.2



**Figure 2: Occupations of women.**

**Table 2: Spirometric measurements of women exposed to ETS at home, workplace and both.**

Parameters	Home (H)	Workplace (W)	(H+W) Both	P value
FEV1 %	1.86	2.35	1.15	0.11
FVC %	2.45	3.03	1.6	0.16
FEV1/FVC	72.89	75.45	70.84	0.99

FEV1/FVC of women exposed at home is 72.89, at workplace is 75.45 and both is 70.84 which is not statistically significant.

## DISCUSSION

During the past two decades deaths from COPD has continued to increase, especially among women.<sup>11</sup> Cigarette smoking is the most important single causal factor for developing COPD. Traffic and other outdoor pollution, second hand smoke, biomass smoke and dietary factors are likely causes of lung function decrement and irreversible airway obstruction. In public health terms, a substantive burden of COPD is attributable to risk factors other than smoking.

To prevent COPD related disability and mortality, effects must focus on prevention and cessation of exposure to smoking and these other less well recognized risk factors. Strong evidence indicates that daily variation in exposure to outdoor air pollution contributes to acute exacerbation of COPD.<sup>12</sup> Present study showed women exposed to ETS at home and workplace had lower FEV1/FVC. A study from China found that self-reported cumulative life time SHS exposure at home and work place was related to greater risk of COPD, as defined by spirometry (GOLD stage 1 or greater).<sup>13</sup> Another study showed that living with a smoker was associated with greater risk of physician diagnosed COPD.<sup>14</sup>

Studies of bar and hospitality workers who were heavily exposed to SHS in the workplace indirectly address the issue of SHS as a possible cause of COPD. After laws prohibiting smoking have been implemented, hospitality workers experienced a substantial reduction in cough and phlegm and an improvement in pulmonary function.<sup>15</sup> Taken together, this evidence supports the plausibility of SHS exposure as a risk factor for COPD. According to recent human studies, even 1 hour of SHS exposure can induce a significant decrease in FEV1 and FEV1/FVC ratio along with cytokine releases, such as interleukins 1 beta, 4, 5, and 6, tumor necrosis factor alpha, and interferon gamma in the lungs, suggesting significant lung inflammation.<sup>16</sup>

As suggested by a few studies, there may be gender, coexisting medical conditions, age or geographical differences in response to ETS exposure, although the reported results were varied.<sup>17</sup> Given this background, this cross-sectional study was conducted to address whether ETS exposure influences lung function adversely

in non-smoking women 20- 40 years of age. Interestingly, we could not find any significant contribution of ETS exposure to lung function. According to our analysis, women who are exposed to cigarette smoke both at workplace and home are more likely to develop COPD later in life. There were some limitations to present study, which should be taken into account when interpreting the results. Firstly, we assessed passive smoking with a questionnaire and secondly, our exposed sample size is small and may have certain implications in interpreting data.

## CONCLUSION

The harmful ingredients of tobacco smoke impact via the blood many organs and tissues causing changes manifested by various symptoms and illnesses. Passive smoking has several subtle as well as overt pulmonary effects. It leads to a poor lung function, small airway dysfunction, and increased bronchial hyper-responsiveness in asymptomatic non-smokers. The problem of passive smoking is complex and represents a serious health hazard that can be prevented by health education campaigns aimed at purveying the evidence about the risk of passive inhalation of tobacco smoke.

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