

# Brief Communication

## Prevalence of active and passive smoking in a population of patients with asthma\*

Prevalência de tabagismo ativo e passivo  
em uma população de asmáticos

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### Abstract

Smoking causes an intense inflammatory reaction in the airways and is associated with worse clinical outcomes in patients with asthma. The objective of this study was to determine the prevalence of active and passive smoking in a population of patients with asthma. The sample of asthma patients (n = 100) consisted of 47 nonsmokers, 33 former smokers, 17 passive smokers and 3 active smokers. Most had moderate or severe asthma. Mean exhaled CO was 9.34 ppb in smokers, 4.19 ppb in passive smokers, 3.98 ppb in nonsmokers and 3.98 ppb in former smokers. We conclude that the prevalence of exposure to tobacco smoke is high among asthma patients.

**Keywords:** Smoking; Asthma; Prevalence.

### Resumo

O tabagismo causa intensa reação inflamatória nas vias aéreas e, em asmáticos, está associado com piores desfechos clínicos. O objetivo desse estudo foi determinar a prevalência de tabagismo ativo e passivo em uma população de asmáticos. A amostra de pacientes com asma (n = 100) consistiu em 47 não-fumantes, 33 ex-fumantes e 3 fumantes ativos. A maioria dos pacientes tinha asma moderada ou grave. A média de CO exalado foi de 9,34 ppb nos tabagistas atuais, 4,19 ppb nos fumantes passivos e 3,98 ppb tanto nos não-fumantes quanto nos ex-fumantes. Concluímos que a prevalência da exposição à fumaça do tabaco é alta entre asmáticos.

**Descritores:** Tabagismo; Asma; Prevalência.

Due to the myriad substances released during the burning of tobacco, smoking directly affects the airways, causing an intense inflammatory reaction. The constant aggression against the respiratory tract due to the daily, cumulative exposure to cigarette smoke causes bronchial hypersecretion and can trigger obstructive diseases, chief among which is COPD.<sup>(1,2)</sup>

The relationship between smoking and asthma was not studied in depth prior to the second half of the 20th century, when the noxious influences of exposure to cigarette smoke in children and adults with asthma, as evidenced by a worsening of the asthma attacks and an increase in the number of new cases of asthma among individuals exposed to cigarette smoke, began to be observed.<sup>(3)</sup>

The World Health Organization estimates that there are approximately 1.25 billion smokers worldwide. Although there is still little information specifically regarding asthma patients who smoke, one group of authors reported that the prevalence of active smokers among asthma patients can range from 17% to 35%.<sup>(3)</sup>

Early detection of active or passive smoking in asthma patients is essential so that smoking cessation programs can be offered to them. Such programs, in addition to decreasing the potential risk for uncontrolled asthma, will principally prevent individuals from developing asthma and COPD in the future.

Therefore, in order to determine the prevalence of active and passive smoking in patients with asthma, we carried out an uncontrolled,

\* Study carried out at the Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo – HC-FMUSP, University of São Paulo School of Medicine Hospital das Clínicas – São Paulo, Brazil.

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open, cross-sectional study. A sample of 100 volunteers was selected from among a total population of approximately 900 consecutive patients regularly monitored at the Asthma Outpatient Clinic of the Pulmonology Department of the *Hospital das Clínicas*, located in São Paulo, Brazil, was evaluated. For all patients, data regarding asthma profile and history of smoking were analyzed, as were data regarding exhaled CO, as measured using a CO meter (Micro Medical Ltd., Rochester, Kent, UK), and simple spirometry data.

Patients were classified based on the criteria for determining asthma severity established by the IV Brazilian Consensus on Asthma<sup>(4)</sup> and divided into current smokers, passive smokers, former smokers and nonsmokers. Smokers were defined as those who reported continuous use of cigarettes, passive smokers were defined as those who lived with an active smoker, former smokers were defined as those who had stopped smoking more than 30 days prior, and nonsmokers were defined as those who were not actively or passively exposed to cigarette smoke. Quantitative data are presented as mean and standard deviation. Only descriptive statistics were used in the analysis of the results.

The study design was approved by the Ethics Committee of the University of São Paulo School of Medicine *Hospital das Clínicas*.

The population studied consisted of 80% women and 20% men. The mean age was 42.7 ± 14.7 years, and the mean number of years of schooling was 6.45 ± 3.74 years. Pulmonary

function characteristics, as well as data related to asthma profile and smoking status, are presented in Table 1.

At the time of data collection, most of the patients reported being nonsmokers or former smokers (47% and 33%, respectively). Only 3% reported being smokers, and 17% reported being passive smokers. Among those who had stopped smoking, 7% continued to be passively exposed to cigarette smoke. The prevalence of active or passive exposure to cigarette smoke was 20% in the sample analyzed.

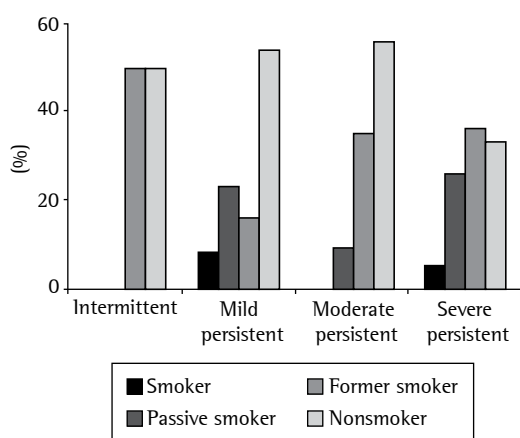
We found that, of the 13 patients classified as having mild persistent asthma, 1 was a smoker (8%), 3 were passive smokers (23%), 2 were former smokers (15%), and 7 were nonsmokers (54%). Among the 46 patients classified as having moderate asthma, there were no current smokers, 4 passive smokers (9%), 16 former smokers (35%) and 26 nonsmokers (56%). Of the 39 patients classified as having severe asthma, 2 (5%) were smokers, 10 (26%) were passive smokers, 14 (36%) were former smokers, and 13 (33%) were nonsmokers (Figure 1).

Our findings differ from those of other similar studies<sup>(3,5,6)</sup> in that the number of smokers in our population was lower. One possible explanation for this discrepancy is the great number of patients in our study who were classified as having moderate or severe asthma. In theory, such patients are more symptomatic and therefore more willing to stop smoking. In one of the studies cited above,<sup>(5)</sup> for example, the mean

**Table 1** – Characteristics of asthma and pulmonary function parameters, by smoking status, in the population studied.

Characteristic/ parameter	Sample as a whole (n = 100)	Active smokers (n = 3)	Passive smokers (n = 17)	Former smokers (n = 33)	Nonsmokers (n = 47)
Type of asthma, n					
Mild intermittent	2	0	0	1	1
Mild persistent	13	1	3	2	7
Moderate persistent	46	0	4	16	26
Severe persistent	39	2	10	14	13
FEV <sub>1</sub> , % <sup>a</sup>	65.5 ± 23.8	61.5 ± 40.3	67.0 ± 23.0	66.7 ± 21.7	64.4 ± 25.9
FVC, % <sup>a</sup>	85.3 ± 21.7	89.0 ± 29.6	89.1 ± 22.5	86.5 ± 20.8	82.8 ± 22.6
FEV <sub>1</sub> /FVC <sup>a</sup>	0.62 ± 0.12	0.56 ± 0.21	0.61 ± 0.11	0.63 ± 0.13	0.63 ± 0.12
Postbronchodilator change in FEV <sub>1</sub> , % <sup>a</sup>	20.3 ± 23.3	6.0 ± 5.6	28.0 ± 26.1	16.4 ± 17.9	21.1 ± 25.2
Exhaled CO, ppb <sup>a</sup>	4.4 ± 3.1	9.34 ± 6.42	4.19 ± 2.90	5.24 ± 4.03	3.98 ± 2.12

<sup>a</sup>Data expressed as mean ± SD.



**Figure 1** – Categories of exposure to cigarette smoke by asthma type.

FEV<sub>1</sub> of the patients studied was  $87.1 \pm 21.1\%$ , compared with  $65.5 \pm 24.1\%$  in our sample.

Our study also showed that, among those who reported being smokers, the mean exhaled CO was higher than that measured in the passive smokers, which, in turn, was higher than that obtained in the nonsmokers. These results are consistent with data in the literature. In one study,<sup>(7)</sup> the authors observed that the measurement of exhaled CO, which is a noninvasive, low-cost method, proved quite useful in evaluating the smoking habit.

Regardless of the degree of asthma severity, 53% of our population reported current or previous exposure to cigarette smoke. This relatively high rate of exposure comes as a surprise, since smoking is a known cause of various respiratory problems.<sup>(6)</sup> Smoking causes decreased pulmonary function even in individuals without a history of respiratory diseases.<sup>(8)</sup> Prolonged exposure can cause bronchitis and emphysema,<sup>(9)</sup> as well as triggering interstitial diseases,<sup>(10)</sup> and is the main risk factor for lung cancer.<sup>(9)</sup>

Curiously, smoking was previously considered part of the treatment for asthma. At the end of the 18th century, studies recommended the use of belladonna alkaloid as the drug of choice for the treatment of asthma attacks. This substance has anticholinergic effects and originates from burning the *Datura stramonium* root, which was found in “asthma cigarettes”, specifically manufactured for the treatment of asthma. The belief that smoking “cured” asthma remained popular until the development of adrenalin in 1930.<sup>(11)</sup>

Within the last 60 years, the noxious effects of exposure to cigarette smoke have been progres-

sively recognized, adverse effects being observed in terms of the development of new cases of asthma and in the worsening of the asthma attacks.<sup>(3)</sup> In a study involving adolescents,<sup>(12)</sup> the development of symptoms suggestive of asthma over a 6-year observation period was found to be associated with three independent risk factors: bronchial hyperresponsiveness to methacholine, atopy and smoking. In another study,<sup>(13)</sup> smoking was considered a risk factor for asthma, especially in individuals over 55 years of age.

In individuals who already have asthma, smoking can negatively affect asthma control and increase the severity of the disease. Studies comparing asthma patients who smoke with asthma patients who do not smoke showed that the former have a greater number of symptoms,<sup>(5)</sup> use a greater amount of relief medication and have a poorer quality of life.<sup>(15)</sup>

The association between smoking and asthma has also been shown to markedly decrease FEV<sub>1</sub>. One group of authors<sup>(16)</sup> reported that the mean decrease in FEV<sub>1</sub> among asthma patients who smoke was 58 mL/year, compared with a significantly lower decrease of 33 mL/year among asthma patients who do not smoke. In a 10-year prospective cohort study,<sup>(17)</sup> the decrease in FEV<sub>1</sub> was found to be 8.5% in nonsmokers without asthma, 10.1% in nonsmokers with asthma, 11.1% in smokers without asthma and 17.8% in smokers with asthma.

Clinical alterations seem to be related to a modification in the inflammatory process that occurs in the airways. Sputum levels of IL-8 are increased in asthma patients who smoke. This presents a positive correlation with the proportion of sputum neutrophils and with tobacco intake (in pack-years), as well as presenting a negative correlation with FEV<sub>1</sub>. Smoking also seems to be responsible for a decrease in the concentration of sputum eosinophils and for alterations in the remodeling of the bronchial mucosa.<sup>(3)</sup>

These pathophysiological alterations partially explain the findings of studies suggesting that the efficacy of corticosteroids is reduced in asthma patients who smoke. A study evaluating the effects that treatment with fluticasone at 1,000 µg/day for 3 weeks has on PEF and FEV<sub>1</sub>, as well as on the provocative concentration causing a 20% fall in FEV<sub>1</sub>, revealed that only the patients who did not smoke presented significant

changes in the values of these parameters.<sup>(18)</sup> Another study, in which inhaled budesonide was used for 9 months, also showed that the asthma patients who smoked were resistant to corticosteroids.<sup>(19)</sup> When prednisolone (40 mg/day) was used, the results were similar.<sup>(20)</sup>

Since there is so much evidence that patient quality of life is worsened—as are clinical control, treatment results and, possibly, the prognosis of asthma—smoking cessation is mandatory in individuals with asthma. Smoking cessation has been shown to have a direct, positive effect, reducing respiratory symptoms such as cough and the production of secretion, as well as reducing the frequency of infectious exacerbations. The speed at which FEV<sub>1</sub> falls becomes once again similar to that found in individuals who do not smoke, and the response to corticosteroids improves, at least partially.<sup>(3)</sup>

In conclusion, the prevalence of exposure to cigarette smoke is high among asthma patients. Therefore, it is extremely important that physicians encourage smoking cessation in asthma patients. Patients who are passive smokers, in turn, should be advised to avoid environments where there are people smoking and to encourage close acquaintances to stop smoking.

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