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Pulmonary Effects of Passive Smoking Among Adults

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Abstract

Passive smoking, also known as environmental tobacco smoke (ETS) or second-hand smoke (SHS) represents the involuntary inhaling of tobacco released by others in the ambient air. Passive smoking exposure occurs in homes, workplaces, and in other public places such as bars, restaurants, and recreation venues. It consists of a complex mixture of mainstream smoke exhaled by smokers and the smoke given off by the combustion of tobacco products. Non-smokers, being exposed to the same toxic substances as identified in mainstream tobacco smoke are, therefore, at an increased risk for serious adverse health effects. Although attention has centered mainly on the harmful effects of SHS exposure in the pediatric population, epidemiologic data from the last 20 years showed increased risks on various respiratory pathologies of the adult. Inhaling SHS causes injury to the respiratory tract, resulting in a high prevalence of respiratory symptoms, asthma, impairment of lung function and increased bronchial responsiveness. In adults, passive smoking is also associated with an increased risk of lung cancer, especially in those with high exposure. On the basis of recent publications, we propose a review of history, biologic basis and effects on different respiratory pathologies of the exposure to SHS in adults.

Keywords: passive smoking, secondhand smoke exposure, tobacco, respiratory diseases, lung cancer

1. Introduction

Tobacco smoke is one of the major health hazards in the general population. Although the large body of research findings has indicated the serious health effects of tobacco in smokers, there is substantial evidence accumulated showing that non-smokers, exposed to tobacco smoke, are also at an increased risk for several chronic diseases, including lung cancer [1, 2].

Passive smoking, also called second-hand smoking (SHS), or environmental tobacco smoke exposure, represents the inhalation of smoke that has been exhaled, or breathed out, by the other person smoking. Most frequent exposure to passive smoking occurs in homes and workplaces [1]. People can be also exposed in public places, such as in bars, restaurants, and recreation venues, as well as in cars and other vehicles [3].

SHS is a complex mixture containing many toxicants, part of them emitted from the smoldering tobacco between puffs, sidestream smoke (SS), and the other part exhaled from the smoker, mainstream smoke (MS) [4]. These components are the same as those that have been identified in MS, over 70 of them being known or suspected human carcinogens [3, 4]. Non-smokers, being exposed at the same toxic substances as identified in mainstream tobacco smoke are, therefore, at an increased risk for serious adverse health effects. Since 1964, more than 2,500,000 deaths among nonsmokers have been caused by exposure to passive smoking [3]. Passive smoking is responsible for significant effects on the health of children, increasing the number and severity of asthma attacks, of respiratory and middle ear infections, and it increases the risk of sudden infant death syndrome [2, 3]. In adults who have never smoked, SHS can cause lung cancer, coronary heart disease, stroke and various respiratory pathologies [1–4]. SHS exposure is responsible for approximately 3000 deaths per year from lung cancer [1]. On the basis of recent publications, this chapter will review the epidemiology, biologic basis, and effects on different respiratory pathologies of the exposure to SHS in adults.

Research reviewed in this article indicates that the level of exposure to SHS has steadily decreased in the United States and most of the European countries over time [5, 6]. This decrease is likely due to the significant declines of smoking prevalence, the growing number of countries with enforced laws to protect non-smoking people against exposure to tobacco smoke (not allowing smoking in indoor areas of workplaces and public places), and the decreased level of the social acceptance of smoking around non-smokers [2, 6].

Despite the large implementation of smoke-free policies, many people are still exposed to tobacco smoke, the level of exposure depending on race and ethnicity, income and occupation, with large differences between different goups [2–4]. In this chapter, the most important actions regarding protection from exposure to second-hand smoke will be discussed in detail.

2. Passive smoking in adults

2.1. Environmental tobacco exposure prevalence

While the literature comprise a large number of statistics on the prevalence of smoking around the world, the magnitude of exposure to SHS among adults, and which socio-demographic groups are at greatest risk, is more difficult to estimate. According to the WHO [7], a majority of the world population (93%) still live in countries not totally covered by smoke-free public health regulations, and exposure to SHS in homes or public places is still common. Globally, among adult non-smoking population, 33% of males, and 35% of females, are regularly exposed to the harmful effects of SHS [8]. The level of exposure varies widely, ranging from 13% in Africa to more than 50% in Eastern Europe or in Western Pacific [7, 8].

Centers for Disease Control and Prevention from the United States reported approximately 58 million (25.3%) non-smokers exposed to SHS in 1 year (2011–2012), down from 52.5% during 1999–2000 [9]. Important differences have been reported between racial and ethnic subgroups, the highest second-hand exposure being encountered among non-Hispanic black (46.8%) compared with non-Hispanic white non-smokers (21.8%) [2]. Data from the European Commission estimated that 28% of European people were exposed to tobacco smoke in bars in 2012, down from 46% in 2009 [6]. In Canada, 27% of nonsmokers self-reported recent SHS exposure [10].

Public places and the workplace were the most frequently self-reported sites of tobacco smoke exposure, but passive smoking was commonly reported in private locations such as personal homes, homes of friends or relatives, and vehicles [2–6].

Tobacco smoke exposure is higher among people with low education and income level. Nearly half (43.2%) of non-smoking adults who lived below the poverty level were exposed to SHS [9]. Disparities in environmental tobacco smoke exposure related to occupation decreased over the past 20 years, but important differences are still recorded. High levels of SHS exposure exist for some groups, including construction an extraction workers, installation maintenance, and transportation workers [9, 11].

2.2. Burden of disease caused by second-hand smoke in adults

Evidence of the harmful effects of passive smoking have been recorded since 1928, when Schonherr suspected that exposure of non-smoking wives to husbands' tobacco smoke, could cause lung cancer [12]. Since then a large body of research findings about adverse health effects of SHS has appeared. The direct link between passive smoking exposure and disability, specific diseases and death has been documented in publication by many international health organizations [1–3, 7]. Furthermore, it has been recognized that there is no risk-free level of exposure to tobacco smoke; even short exposures can be harmful to health [2, 13]. The health effects attributable to SHS in adults have been quantified as deaths and disability-adjusted life-years (DALYs) lost [7, 8].

Passive smoking was estimated to have caused 603.000 deaths in 2004, corresponding to 1.0% of worldwide mortality [7]. Of these, 35.800 deaths were attributed to asthma and 21.400 to lung cancer that occurred in non-smoking adults. As a result of deaths caused by tobacco exposure, 6.6 billion dollars were lost in productivity [14]. More deaths attributable to SHS exposure, with 281.000 (47%) per year, occurred in women, compared with 156.000 (26%) in men [7, 8]. This situation could be explained by the fact that the absolute number of non-smoking women is approximately 60% higher than that of non-smoking men [13]. Second, in many parts of the world, such as Africa, the Eastern Mediterranean, Southeast Asia, and some parts of the Americas, women are at least 50% more likely to be exposed to passive smoking than are men [8].

The evaluation of the burden of disease, quantified as DALYs lost because of exposure to SHS, revealed a number of 10.9 million DALYs lost that occurred worldwide, in 2004, corresponding to 0.7% of the total worldwide burden of diseases in DALYs [7, 8]. A significant part (1.246.000) was due to asthma in adults.

Considering all these adverse health effects, passive smoking is considered to be the third most frequent risk factor for avoidable deaths, after active smoking and inadequate dietary intake [15, 16].

3. Respiratory effects of passive smoking in adults

Passive smoking causes many of the same diseases such as direct smoking, including lung cancer, respiratory and cardiovascular diseases [1–5]. This chapter focuses on the respiratory health outcomes of involuntary smoking in adults, such as respiratory symptoms, pulmonary function, and respiratory diseases (**Table 1**), detailing chronic obstructive diseases and lung cancer.

3.1. Pathophysiology

Second-hand smoke is formed mainly (85%) of sidestream smoke (SS), which is the product of incomplete combustion, released from the burning tip of a cigarette, and only a small part (15%) of mainstream smoke (MS), which is exhaled by smokers [17]. The diameter of SS components is 10 times smaller compared to the particle diameter of MS and, as such, SS components have the potential to reach the most distal alveoli from where they cannot be expelled easily [17, 18]. The composition of environmental tobacco smoke consists of a complex mixture of more than 4000 chemical substances, with proinflammatory and cytotoxic effects [19].

Several experimental studies conducted on animals [20–22] and humans [23, 24] showed relevant evidence and information on the underlying mechanisms for the effects of passive smoking on the respiratory tract. Pulmonary emphysema, including the loss of elasticity in the lung tissue, was induced in rats through a 3 months exposure to tobacco smoke (mean CO at 35 ppm) for 90 min per day [20]. Alterations of airway defense mechanisms and enhanced allergic inflammatory responses has also been observed as a response to SS exposure [21, 22]. High levels of total serum immunoglobulin E (IgE) were observed in adult population exposed to cigarette smoke [25], although not all studies found this association [26]. IgE enhanced values might be associated with the later development of allergies [27]. The increase in susceptibility to allergic diseases may also be explained by the depletion of Thl cytokine-secreting cells in the human airway caused by cigarette smoke exposure [22].

The cigarette smoke induces increases of neuroendocrine cells in the lung, which synthesize and release bronchoconstriction mediators, responsible for bronchial hyperreactivity and asthma [26]. The stimulation of C-fibers by components of SHS including nicotine, acrolein,

Respiratory symptoms	Respiratory illnesses	Pulmonary function
Cough	Asthma	Declines in FEV1 levels
Phlegm production	COPD	Increased bronchial responsiveness
Wheeze	Exacerbation of cystic fibrosis	
Chest tightness	Allergies	
Dyspnoea on exertion	Bronchitis	
Shortness of breath at rest	Pneumonia	
Nocturnal breathlessness	Bronchiolitis	
	Lung cancer	

Table 1. Environmental tobacco smoke-related respiratory effects in adult population.

and oxidants can trigger intense respiratory responses through local and central nervous system reflexes, inducing mucous secretion, bronchoconstriction, and increased microvascular leakage [28]. In vivo and in vitro studies have demonstrated the inhibitory effect of cigarette smoke on exhaled nitric oxide (NO) [24, 29]. The inhibition of NO synthesis increases bronchial response in persons exposed to SHS [24]. Bronchial hyperreactivity may also result from smoke-induced inflammation [24, 29]. The outcome of several studies showed an increase in circulating PMNs, significantly greater PMN chemotactic activity, and the augmented release of oxidants upon stimulation, in nonsmokers exposed to SHS [23, 30].

Exposure to passive smoking increases susceptibility to respiratory infections and/or worsens infections through the inhibition of antibody responses, impairing macrophage responsiveness and mucociliary clearance, increasing bacterial adherence, and disruption of the respiratory epithelium [31, 32].

3.2. Non-malignant respiratory health effects

In the last years, there has been published more information regarding the causal relationship of passive smoking with acute and chronic respiratory health effects in adults.

3.2.1. Respiratory symptoms

In several large cross-sectional population studies, SHS exposure was found to be an independent risk factor for different respiratory symptoms, including wheezing, cough, dyspnea, nocturnal breathlessness and shortness of breath at rest [26, 33–36]. The outcome of all studies strengthened the evidence of a causal effect by a significant dose effect relationship between the amount of smoke exposure and the possibility of having symptoms. Among different irritants, such as car exhaust fumes, cold air and strong smells, passive smoking was the most often identified by the subjects as causing lower airway symptoms [34]. Data from a study involving 2335 never-smoking Italian women, of which, 73% reported lifetime SHS exposure, showed that the combined exposure both to the husband and at work was a significant risk factor for each health outcome. Current dyspnea was the most frequently (26.9%) reported health outcome [36]. A recent study has provided the evidence of significantly increasing the risk of persistent respiratory symptoms into young adult life if there was exposure to parental smoking during childhood [35].

3.2.2. Lung function and bronchial responsiveness

Studies of the relation between passive smoking and lung function have generally found a harmful effect of such exposure [26, 33, 37–39]. The extent of this impairment is, however, relatively low, with a decrease of forced expiratory volume in 1 s (FEV1) in the range of 50–100 mL in subjects with the highest level of exposure compared to unexposed subjects [37]. Some studies have shown that passive smoking is associated with decreased lung function suggestive of airflow limitation (FEV1 and FEV1/FVC) in female but not male non-smokers [33, 38]. Women with middle level exposure had on average 88 mL reduction of FEV1 and 2% for FEV1/FVC compared to those with low exposure [33]. This gender-specific susceptibility of lung function impairment in women can result from the fact that most studies usually

involve a small number of men non-smokers. The results of the European Community Respiratory Health Survey (ECRHS) involving a large adult population (18.992 subjects) from 17 European countries, have demonstrated a positive association between exposure to parental smoking in childhood and impaired lung function in adult lifetime [39]. Regarding the short-term passive smoking effect on lung function in asthmatic adult population, the report of the Surgeon General concluded that the evidences are suggestive but not sufficient to infer a causal relationship [40]. The impairment of pulmonary function among asthmatic adults, particularly in asthmatic women, was demonstrated in The Swiss Study on Air Pollution and Lung Diseases in Adults (SAPALDIA), where the duration of environmental smoke exposure at work was associated with a decrease in lung function (FEV1–6% per hour of smoke exposure at work ($p = 0.01$); FEF25–75%: -3.4%/h ($p < 0.05$)) [41].

Association between passive smoking in adulthood and increased bronchial responsiveness has been shown in both epidemiological [26] and experimental studies [42, 43]. Adults with asthma have even greater bronchial hyperresponsiveness after SHS exposure [39, 44].

3.2.3. Asthma

A large number of published studies have examined the links between involuntary smoking and new-onset asthma and the exacerbation of pre-existing asthma among adults [10, 19, 33, 36, 38, 41, 44–54]. The causal relationship between SHS exposure and self-reported or physician diagnosis of asthma has been observed in a large variety of study designs, including case–controlled, cross-sectional, and cohort studies, involving different populations from different countries around the world [45].

Passive smoking exposure during childhood has been shown to lead to an increased prevalence of asthma diagnosis in never-smoking adults, especially those without any family history of asthma [19]. In a large population-based Swedish study involving over 8000 never-smoker adults, the prevalence of physician-diagnosed asthma was more frequently reported among subjects who reported environmental tobacco smoke exposure during childhood (7.6%), compared with unexposed persons (5.8%) ($p = 0.035$) [46]. Another study also found a similar pattern of results, with a 1.39-fold increase of asthma diagnosis in the never-smoking adults with childhood exposure to passive smoking [47].

Numerous studies assessed the causal relationship between the whole lifetime exposure to passive smoking and new-onset asthma in adults. Increased risk of asthma was observed among non-smokers adults exposed to passive smoking home only [48–50], at work only [51, 52], and to both types of exposures, home and workplace [10, 33, 36, 41]. Home exposure to parental smoking was related to an increased risk of self-reported asthma in young adulthood (OR 1.8; 95% CI 1.1–3.0 for maternal smoking, OR 1.6; 95% CI 1.1–2.4 for paternal smoking, respectively) [48]. The results of another population-based case–controlled study have also demonstrated the strong association between home exposure to passive smoking and adult-onset asthma but with a higher risk among males (OR 4.8; 95% CI 2–11.6) than females (OR 1.5; 95% CI 0.8–3.1) [49]. An Indian population-based cross-sectional study involving 62,109 never-smoking adults showed a higher risk of having asthma among subjects who reported both childhood and home exposure (OR 1.69; 95% CI 1.38–2.07) [50].

Regarding workplace exposure, it has been shown that daily exposure to passive smoking (more than 5 h) increases the risk of physician-diagnosed adult asthma (OR 1.79) [34]. The duration of working with a smoker is directly related with an increased risk of developing asthma (OR 1.5 per 10-year increment; 95% CI 1.2–1.8) [51, 52], but a population-based case-controlled study from Finland has shown a strong association between new-onset adult asthma and passive smoking exposure during only the preceding 12 months of asthma diagnosis (OR 1.97; 95% CI 1.19–3.25), with evidence of an exposure response relationship (OR 1.33 per 10 cigarettes per day; 95% CI 1.02–1.75) [53].

Current evidence suggest that passive smoking in asthmatic subjects is related to a negative effect on prognosis, with an increased risk of exacerbations and hospitalization, pulmonary function impairment, and a decrease in the quality of life [37, 45]. Extended data from literature, including studies [10, 36, 38, 41, 44, 54] and reviews [37, 40, 45] strongly link passive smoking with poorer asthma status in adults diagnosed with this condition.

3.2.4. Chronic obstructive pulmonary disease

Compared to asthma, the causal association between passive smoking exposure and chronic obstructive pulmonary disease (COPD) has been less widely the object of epidemiological studies [16, 55–59]. The relative risk (RR) for the association between SHS exposure and COPD, calculated in a recent published meta-analysis [60], based on cohort and case-control studies (shown in **Table 2**), was 1.66, with a comparatively small confidence interval (95% CI: 1.38–2.00). A higher RR of 2.17 (95% CI: 1.48–3.18) was obtained for women, than for men, of which RR of 1.50 (95% CI: 0.96–2.28) was inherited from one single study [55].

In a large population-based study conducted in Germany [16], whose of aim was to quantify the adverse health effects attributable to passive smoking, the results indicated that 231,973 persons were affected by COPD caused by SHS exposure in 2014, representing 0.35% of the male and 0.22% of the female German population. The higher prevalence of COPD cases attributable to passive smoking occurred in the age group 60 years and older (1.15% of men and 0.55% of women).

Similar to the impact of passive smoking on asthma evolution, several studies have shown the worsening effect of SHS exposure on COPD patients [56–58]. In longitudinal analysis, the highest level of tobacco exposure, measured by cotinine urine level, was associated with worse COPD severity [56]. Another publication observed an increased risk of dying from COPD in never-smokers exposed to SHS [57]. Passive smoking in COPD patients was also associated with increased hospital readmission rates [58].

3.3. Lung cancer

A major shift in policy approaches to controlling tobacco occurred in the late 1980s, with the rise of new evidence that SHS causes death and disease in non-smokers [59], especially after 1981, when Hirayama's study demonstrated that non-smoking wives of smokers were almost twice as likely to die of lung cancer compared with non-smoking wives of non-smokers [68]. In 1986, the US Surgeon General, the International Agency for Research on Cancer, and the US

Authors	Type	Participants	Exposure	Relative risk (95%CI)
Kalandidi et al. [61]	Case–control	103 female patients, 179 female controls, never-smokers	home: spousal smoking (1–20 cigarettes/day)	women: 1.79 (1.17–2.57)
McGhee et al. [55]	Case–control	4838 cases (2680 men, 2158 women) 763 controls (418 men, 345 women), never-smokers	home	1.81 (1.24–2.65) men: 1.50 (0.96–2.28) women: 2.59 (1.30–5.27)
Chan-Yeung et al. [62]	Case–control	289 patients (243 men, 46 women) 289 controls (243 men, 46 women)	home and workplace	1.64 (0.97–2.03)
Yin et al. [63]	Cohort	15,379 (1777 men, 13,602 women) never-smokers	home: ≥ 5 years of 40 h/week	1.60 (1.23–2.10)
Schwartz et al. [64]	Case–control	562 female cases, 564 female controls	home	women: 1.68 (1.12–2.61)
Wu et al. [65]	Case–control	205 female cases 205 female controls	home and workplace	women: 3.12 (1.56–6.50)
Johannessen et al. [66]	Case–control	433 patients (258 men, 175 women) 325 controls (176 men, 149 women)	home	men: 0.98 (0.81–1.17) women: 1.14 (0.93–1.37)
He et al. [67]	Cohort	910 (439 men, 471 women) never-smokers	home and workplace	2.30 (1.06–5.00) men: 2.15 (0.86–5.39) women: 3.31 (0.69–15.82)

[1]Adapted from Fischer F et al. [60].

Table 2. Studies assessing the relative risk for the association between passive smoking exposure and COPD[1].

National Academy of Sciences concluded that SHS causes lung cancer in non-smokers [40]. Environmental tobacco smoke has been classified as a known (Group A) human lung carcinogen by the United States Environmental Protection Agency (EPA), since 1993 [2]. After this year, this risk factor was intensively studied in relation to lung cancer. At least 17 carcinogenic chemicals contained in tobacco smoke are emitted at higher levels in sidestream smoke than in mainstream smoke [40], with one like benzo(a)pyrene diol epoxide, which shows a direct etiological association with lung cancer, founded also in both mainstream and side stream smoke [69].

SHS exposure is associated with an excess relative risk of lung cancer of around 20% [70]. The results of the meta-analysis published in 2007 have shown a 27% excess in the risk of

lung cancer among never-smoking women exposed to spousal passive smoking, compared with not exposed never-smoking women [71]. Exposure at home seemed to have a stronger effect than exposure at the workplace, probably because exposure at home, especially from the spouse, is more likely to be of greater duration and intensity than exposure at work. Other results suggest that people with combined exposure (home and work) are more likely to develop lung cancer than those exposed at one location only [70, 72].

Specific and nonspecific pathways through which smoking causes cancer are common for passive and active smoking. There are DNA binding and mutations, but also epigenetic mechanisms. Tobacco-specific carcinogens form adducts and lead to mutations in oncogenes and tumor-suppressor genes [73].

A prospective study in 10 European countries, estimated the proportion of lung cancers in never- and ex-smokers attributable to passive smoking at 16–24%, mainly due to the contribution of work-related exposures [74].

On the overall population, exposure to SHS smoke increases the risk of lung cancer by approximately 30 and 60% for non-small cell lung cancer (NSCLC), and small cell lung cancer (SCLC), respectively [75]. Among never smokers, passive smoking increased the risk by approximately 30 and 200% for NSCLC and SCLC, respectively [74, 75].

Lung cancer in never smokers has been recognized as a distinct clinical entity apart from lung cancer in former/current smokers due to the initial observation of a significantly higher prevalence of adenocarcinoma (ADK), female gender, and advanced stage at presentation, but a better overall survival compared with lung cancer in ever smokers [76]. There is weak evidence that exposure to SHS increases the risk of lung adenocarcinoma in situ/minimally invasive adenocarcinoma [77].

The adjusted ORs for the association between passive smoking and lung cancer among never smokers were 1.26 (95% CI 1.10–1.44) for ADK, 1.41 (95% CI 0.99–1.99) for squamous cell carcinoma, 1.48 (95% CI 0.89–2.45) for large cell carcinoma, and 3.09 (95% CI 1.62–5.89) for SCLC [75].

There is no clear association between passive smoke exposure and somatic profile in lifelong, never-smoker lung cancer, even if EGFR and HER2 mutations and ALK rearrangement are known to be related to lung cancer in never-smokers, while KRAS, BRAF and PIK3CA mutations are typically observed among smokers [78].

4. Protection from exposure to second-hand smoke and impact on respiratory diseases

After 2005, when the Framework Convention on Tobacco Control (FCTC) has been acceded to, ratified, accepted, or approved by 40 States, many governments had a multitude of possibilities of interventions at their disposal to reduce tobacco use and SHS exposure, such as raising cigarette taxes, banning advertising and promotion of tobacco products, creating smoke-free areas in public places and worksites, mandatory introduction of health warnings

on cigarette packs, and promoting educational mass media campaigns regarding the harmful effect of tobacco use. From that moment, despite the efforts, the differences in implementation of FCTC measurements are still persisting from one country to another [79, 80]. Nevertheless, smokers in EU countries with high tobacco control scale (TCS) scores are more concerned about the effect of their smoke on others; this can be regarded as a marker of tobacco use denormalization, and as a support for tobacco control measures. These findings support the idea that the issue of passive smoking is essential for tobacco control in Europe [80]. It also happens like this for other continents. Within 20 years of the first Surgeon Generals' Reports, research showed that even the far lower doses of SHS have harmful effects on respiratory health [81].

4.1. Smoke-free legislation and his consequences

Self-regulation and smoking restrictions imposed by individual employers and venue operators have failed to protect staff and patrons in many enclosed environments, such as restaurants, pubs and casinos [82].

Smoke-free legislation is the best way to protect the passive smokers from SHS. Those who benefit most from this kind of policy are, undoubtedly, non-smokers; tobacco smoke is eliminated from the workplace, and they are able to breathe better-quality air. In addition, the working environment is improved thanks to the existence of a consensus among all the workplace's employees, which may lead to the resolution of possible conflicts between smokers and non-smokers [83]. Lack of knowledge of the health consequences of passive smoking exposure is an information failure and an important motivator of smoke-free policies.

Extended data from literature have described the tobacco industry's efforts around the world to undermine and discredit the evidence-based link between passive smoking and disease [84, 85]. Comprehensive smoke-free measures are the only effective means of fully protecting the public from the risks associated with SHS exposure. Partial measures that restrict, rather than eliminate, indoor smoking (e.g., designated smoking sections, separate ventilation) can reduce the exposure but do not offer adequate protection from his harmful health effects [86]. The offers of tobacco industry and tobacco lobbyists, such as "separating smokers from non-smokers, cleaning the air, and ventilating buildings, cannot eliminate exposure of non-smokers to second-hand smoke," as the US Surgeon General has concluded [40].

At the end of 2014, 49 countries had implemented the national comprehensive smoke-free legislation. This means that 18% of the world's population (1.3 billion people) is protected from the dangers of SHS by a comprehensive smoke-free law [87]. WHO reports that strong smoke-free legislation is the most widely adopted tobacco control measure, but low-income countries are less likely to have it adopted, than high- and middle-income countries [88]. In a growing number of countries, smoke-free policies have been extended beyond workplaces and indoor public spaces to include previously unregulated areas, such as outdoor spaces (public beaches and in public parks, as in Vancouver, or in pedestrian areas, as in New York) [87].

Comprehensive smoke-free legislation is a major policy intervention that works at several levels. It improves air quality and reduces non-smokers' SHS exposure, encouraging smokers

to reduce their tobacco consumption, limiting the times and places where they can smoke and motivating smokers to attempt to quit [89].

Smoke-free policies improve health outcomes. We have now significant and consistent evidences from around the world, showing that comprehensive smoke-free laws are associated with improved respiratory health and reduced cardiovascular disease [90–96]. There are multiple examples: 1 month after the implementation of a smoke-free law in Scotland, asthmatic bar workers demonstrated improvements in airway inflammation and self-reported quality of life [90], a smaller decrease in lung function was observed from the beginning to the end of a work shift, after smoke-free legislation was implemented in Norway [91], less likely to report adverse symptoms such as wheezing, coughing, and shortness of breath in the months after that state's smoke-free law took effect in hospitality workers in New York State [92], fewer current asthma symptoms and fewer physician visits because of asthma symptoms [93], and reduced asthma incidence, symptoms, and hospitalizations and/or emergency room visits in adults and/or children [94].

The lowest levels of SHS exposure are found in workplaces that completely ban smoking. Completely smoke-free workplaces achieve a number of public health benefits, such as reducing workers' exposure to the toxins contained by cigarette smoke, by 80–95% [95].

Regarding home SHS exposure, it has been observed that people of lower individual socioeconomic status are less likely to have smoke-free home rules and less likely to be protected by smoke free workplace policies [96], and that is why future SHS interventions need to pay more attention to people with this condition, in order to gain a better control of SHS exposure.

4.2. Other tobacco control measures

A 50% increase in cigarette prices will correspond to a 30–40% decrease of tobacco use for the poor, a much larger relative decline than among the rich. Another major consequence of this measure is that poor people benefit the most, economically and in health, from smoking cessation following a tax rate hike [88]. Evidence from Thailand showed that the poor paid only 6% of increased tobacco taxes, but got 58% of the health benefits [97]. These benefits consist of the consequences on respiratory and cardiovascular diseases, not only for smokers but also for nonsmokers. Taxes are very important; an increase of 10% in the price of cigarettes would decrease cigarette consumption by 3–5%, and, if implemented comprehensively, it could prevent 5–16 million smoking-related deaths worldwide and SHS exposure, also [98]. A number of studies have demonstrated that youth are relatively more sensitive to price than adults, implying that raising cigarette taxes would be a useful tobacco prevention intervention [88, 97, 98].

The major information source regarding awareness on the health effects of SHS exposure was radio media advert, especially in low income countries. Multi-level tobacco-control strategies should therefore include messages about harmful effects of SHS exposure to protect innocent never-smoked individuals, while warnings on cigarette packets should also include information on the deleterious effects of SHS on the health of adult, children, and pregnant women [99].

Educational activities are also important. It is a lack of appreciation among the general population of risk posed by SHS exposure to a vulnerable subset of population, namely,

children. Protection of children from SHS needs global attention and special educational campaigns in schools but also in adult population [100]. Social diffusion posits that restricting smoking in public spaces leads to increases in voluntary home smoking bans or restrictions [101]. Smoke-free legislation may stimulate smokers to establish total smoking bans in their homes.

An introduction of pictorial warning labels was shown to have a statistically significant effect on smoking prevalence, exposure of non-smokers, and quit attempts and significantly decreased the odds of being a smoker. Plain packaging of tobacco introduced in 2012 in Australia, brought a 78% increase in the number of calls to the national cessation quit line [102]. It is another action with a proof impact.

For electronic nicotine delivery systems (ENDS) and Heat-Not-Burn (HNB) tobacco products, governments should ban indoor use, because the aerosol released from both tobacco products contains many of the harmful constituents found in cigarette smoke [103]. There are more studies in this moment for e-cigarettes. There are likely health risks from being exposed to second-hand aerosol of HNB tobacco products [103, 104], but long term studies for analyzing the second hand effects at a distance are needed.

Policy and advocacy with effect on SHS concerning respiratory diseases must be complex and should use multiple action tools. In line with the FCTC, in 2008, WHO introduced the MPOWER measures, consisting of a set of six cost-effective and high-impact measures that help countries reducing the demand for tobacco and also for SHS exposure. These measures consist of: "monitoring tobacco use and prevention policies, protecting people from tobacco smoke, offering help to quit tobacco use, warning about the dangers of tobacco, enforcing bans on tobacco advertising, promotion and sponsorship, and raising taxes on tobacco." At the moment, more than half of all countries, representing approximately 40% of the world's population, have implemented at least one MPOWER measure to the highest level of achievement [87].

5. Conclusions

Passive smoking is a major wide-spread contaminant of indoor air and represents an important risk factor for pulmonary diseases among adult population. Extent epidemiologic evidence showed the strong association between passive smoking exposure and persistent respiratory symptoms, impaired lung function, and bronchial hyperresponsiveness. New-onset asthma and COPD, along with worsened prognosis of both diseases, are also attributable to passive smoking. Among never smokers, passive smoking increases the risk by approximately 30 and 200% for NSCLC and SCLC, respectively. Policies prohibiting smoking in the community have multiple positive effects on respiratory health. Comprehensive smoke-free legislation is a major policy intervention that should be widely implemented to protect non-smokers from the adverse health effects caused by passive smoking.

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